JULY, 1952

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Histochemistry of the Gastrointestinal Tract

Spontaneous Rupture of the Esophagus

Surgical Lesions of the Esophagus

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Seventeenth Annual Convention

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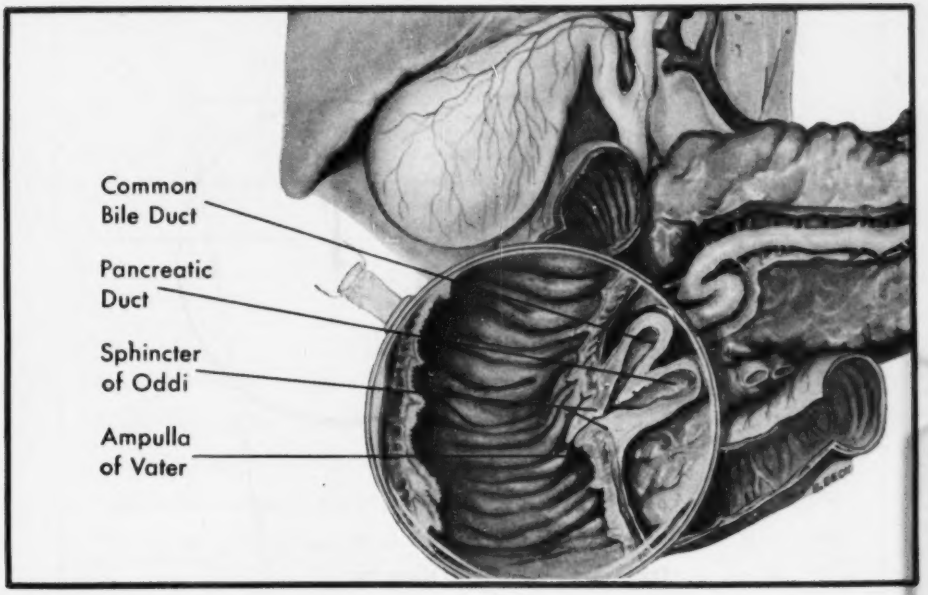

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

VOLUME 19 JULY, 1952 NUMBER 7

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Index to Advertisers

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OFFICIAL PUBLICATION

of the

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

1819 Broadway, New York 23, N. Y.

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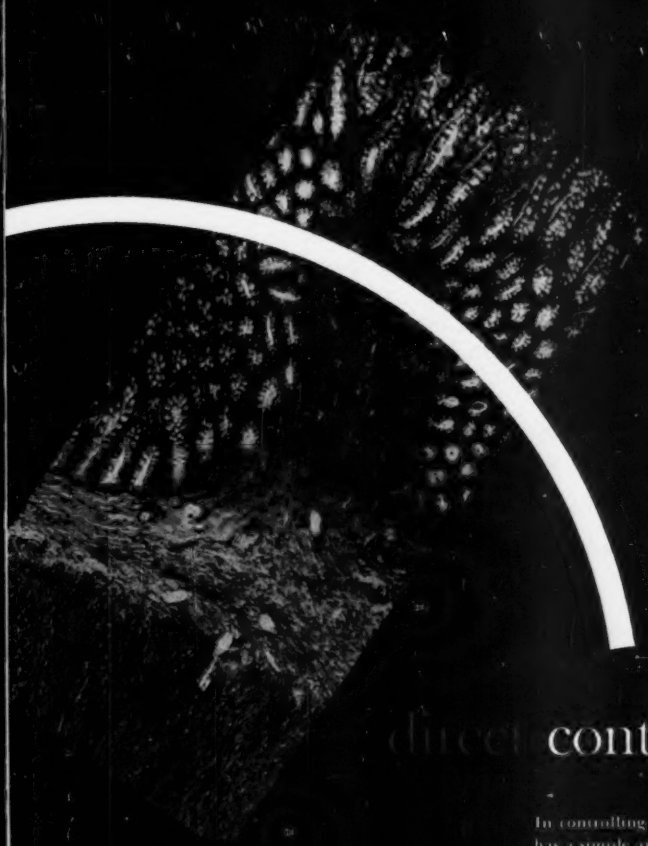

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Communications regarding business matters, advertising, subscriptions and reprints should be sent to the Business office, 1819 Broadway, New York 23, N. Y.

Subscription price: U.S. and Pan-American Postal Union: one year, $5.00, two years, $9.00. Elsewhere, $7.00, $13.00. Single copy $.50. Members of the National Gastroenterological Association receive the REVIEW as part of their membership.

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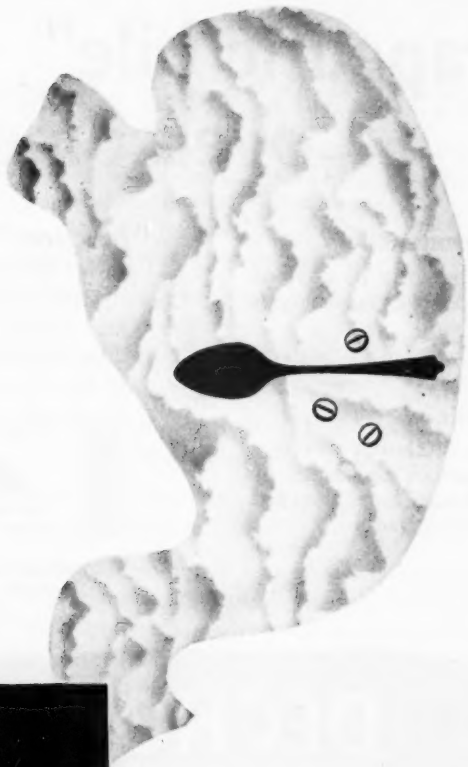

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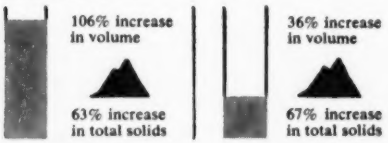

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The Review of Gastroenterology

A monthly journal of Gastroenterology, Proctology and Allied Subjects
(Incorporating the American Journal of Gastroenterology)

Volume 19 JULY, 1952 Number 7

SURGICAL LESIONS OF THE ESOPHAGUS*†

Including Discussions of Esophagitis, "Spontaneous" (Acid-Peptic) Perforation of the Esophagus, Dystonia (Cardiospasm) Paraesophageal Hernia, Varices and Cancer, Together with Remarks Upon Surgical Management of Peptic Ulcer.

OWEN H. WANGENSTEEN, M.D., F.A.C.S.
Minneapolis, Minn.

The preceding discussion has interested me, as a surgeon, very much. We surgeons, though we affect an air of independence, we too, like all clinicians are very dependent upon other colleagues. Moreover, it is necessary for us to confess often to ourselves, if not to others, how fallible we can be.

Now, the subject that I have chosen for discussion today is essentially one, which might, in the minds of many, come very properly into the area of interest of the psychosomaticist or possibly the psychotherapeutist. However, I think I shall be able to show you that, such lesions as esophagitis, "spontaneous" perforation and esophageal dystonia are essentially organic problems .

I have been looking at these entities through two windows! One that of an observer – a surgeon, who sees ready-made experiments in hospital beds and in the out-patient clinic, and the other, as an experimentalist striving to analyze and interpret what I have observed.

The whole story of many esophageal lesions, of course, is wrapped in mystery. Great need exists for more factual information which will afford better clarification of some of these disorders.

Cancer

I would like to begin this discussion, which of necessity, in many areas of interest, must be somewhat fragmentary, in reverse order, by a brief reference to

*Read before the Sixteenth Annual Convention of the National Gastroenterological Association, Chicago, Ill., 17, 18, 19 September 1951.

†This presentation is based upon researches supported by the following funds for Surgical Research: 1. Institutional Grant, American Cancer Society; 2. Austen S. Cargill Fund; 3. Augustus L. Searle Fund; 4. Jay and Rose Philips Fund and 5. Mr. and Mrs. R. C. Lilly Fund.

the surgery of esophageal cancer. Professor Ferdinand Sauerbruch of Berlin, who died recently, wrote several interesting and fascinating books. He established in his monographs on thoracic surgery a prototype for publication which we, in America unfortunately, have yet not been able to emulate. The beautiful illustrations in color, the quality of the paper, the scholarship which he brought to the effort – all these make you feel that you hold in your hand, not only a beautiful tome but one which exacted much labor, and one which brought together many great and varied talents in its composition. Yet, when one reads those books critically now, it is amazing to note how little Professor Sauerbruch had to say about the therapy of esophageal lesions. In fact, it seems somewhat strange to us now that Sauerbach, despite all his book-learning and making and his large personal experience in the perfórmance of operations upon the esophagus, made no important contribution to the surgical management of lesions of the esophagus. It remained for Oshawa[1] in far off Kyoto, Japan, to break through the darkness and fog of failure which surrounded this problem and to bring hope, light and order to the surgical problem of effecting restoration of continuity after surgical excision of segments of the esophagus for cancer. By a few carefully analyzed experiments, and by the simple expedient of discarding completely the previous emphasis of need for hurry in the execution of the operation, Oshawa was able to succeed in excising the lower esophagus for cancer, with reestablishment of esophagogastric continuity, where more experienced surgeons had failed previously.

The literature of esophageal surgery is full of tragedy. Billroth (1881) succeeded the first time he undertook excision of a cancer of the pylorus in man. And, how long surgeons were denied operative success in attempts at excision of cancer of the thoracic esophagus with full restoration of continuity of the alimentary tract! Torek (1913) was able to report the successful removal of a cancer of the thoracic esophagus with the establishment of a cervical esophageal fistula, some semblance at an effort of restoring continuity being effected by the insertion of a rubber tube between esophagostomy and gastrostomy openings. And how many times similar successes were denied other surgeons, who employed Torek's method! In their monograph, of 1926, on Surgery of the Esophagus, von Hacker and Lotheissen[2] state that, more than 100 such operative attempts had been reported by 30 surgeons with none, other than short-lived successes. Some surgeons, confusing hope with achievement, even felt constrained to report as successes brief survivals of 8, 14 and 24 days! And yet, intermittently, surgeons continued this frontal assault upon the problem of cancer of the thoracic and subdiaphragmatic esophagus until the technical problems of the operation were resolved satisfactorily. Oshawa (1933) showed the way, and now surgeons, everywhere, succeed quite regularly with reasonable mortality rates, where previously, uniform failure was the rule. And the surprising thing is that, operations proposed by Kirschner[3] (1920), and others are virtually the exact prototype of today's successful proced-

ures. Surgery is replete with anachronisms; the surgery of that day was not ready for the problems, which operative undertakings of that magnitude presented. Surgeons succeed today with the difficult problems of esophageal surgery where their forbears failed because now information has been synthesized out of old facts. The appraisals are better; the technics are very much the same, but the emphasis is a little different and the integrations are far better. Perhaps no sorry chapter in all of surgery carries a more instructive lesson. The more important problem now is to learn to recognize the presence of a cancer of the esophagus early enough so that successful excision will more regularly be followed by a lasting cure.

Satisfactory technics have now been worked out for every cancer of the esophagus, save those of the hypopharynx. Surgeons everywhere have accepted the thesis that unless continuity can be reestablished, the operation for esophageal cancer should probably not be done. Only in cancers of the hypopharynx is it still necessary to establish a temporary or permanent fistula. For some of these, shortening of the path for the mobilized and thoracically migrated stomach may do away with that unacceptable feature of the procedure. On first glance, it may seem a bit far-fetched to employ the stomach to supplant the cervical esophagus. It would seem there should be simpler means of making a tube for the upper esophagus. The truth of the matter is, however, that only the stomach can be migrated up in a one-stage procedure. To be certain, the jejunum could be used by cutting some of the jejunal vessels of the segment to be swung upward, but it is not safe to attempt this in one procedure. And all the present-day procedures employed to replace a segment of the cervical esophagus with skin grafts, or skin grafts reinforced with a prosthesis, must be done in stages, and at the risk of temporary or persistent fistula.

In a few patients with cancer of the upper thoracic esophagus, I have employed a sternotomy incision[4], splitting the sternum in the mid-line from the ensiform process into the right first interspace. Below, the incision is carried to the umbilicus. The right diaphragm is split down to the vena cava. The esophagus is mobilized and is severed from the stomach on the distal end of the esophagus. The mediastinal pleura to the right of the heart is opened and the esophagus is delivered into the right thorax below the pulmonary vein to the lower lobe of the right lung. At this juncture, the patient, who up until now has been lying supine on the operating table, is inclined to the left, bringing the right thorax up to a convenient angle. The draping is not disarranged by this maneuver. The first assistant now holds the lung forward and the surgeon frees the esophagus from its surrounding structures. By employing the silver-clip device employed by neurosurgeons for catching the vessels, perfect hemostasis can be obtained. Even an adherent cancer can be mobilized safely and under direct vision by this technic. When the esophagus has been mobilized a little beyond the upper root of the lung, the patient is returned to the supine position on the operating table, the azygos vein is divided and the dissection is continued. The stomach is brought up

just to the right of the heart, in front of the lung. The pylorus, after adequate mobilization of the duodenum comes now to lie at or above the diaphragm.

I believe, the surgeon, accustomed to the left thoracic approach, who employs this method, will find himself excising a longer segment of the esophagus proximal to the cancer – a procedure distinctly advantageous from the standpoint of cure. Up until now, in any case, I have had no opportunity to do this procedure save for lesions of the thoracic esophagus. By closing the diaphragmatic hiatus and bringing the esophagus up just over the vena cava, it is astounding how far up the mobilized stomach will reach. I have the feeling that it may be possible to bring the fundic end of the stomach, in this manner, to the level of the pharynx fairly regularly in one stage. For an anastomosis at that level, it might prove advantageous to swing a mobilized gastric tube with an antral flap uppermost – not in the interest of gaining length necessarily, but because that segment contains no acid secreting cells. Despite the circumstance that such a mobilized stomach is vagotomized, its parietal acid-secreting cells still function actively, and it probably would not be pleasant to have that fundic portion of the stomach at the very base of the floor of the mouth. Obviously, the cutting of such a flap would have to be a staged procedure, to insure satisfactory vacularization of the flap.

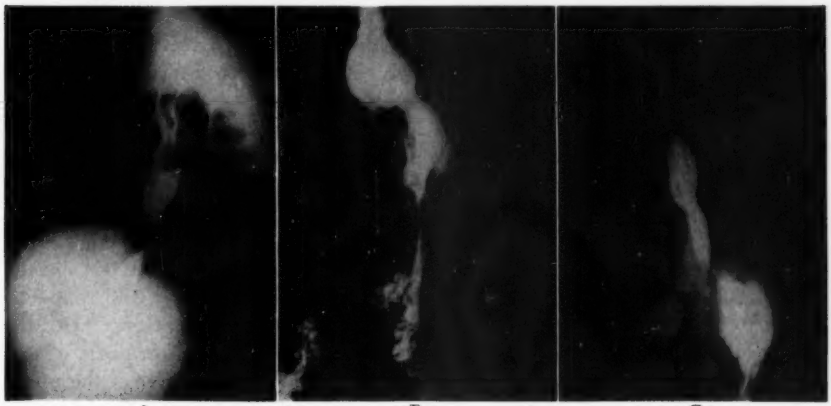
I have toyed with the idea of severing the remaining gastric vessels near the pyloric end of the stomach, upon which the migrated stomach hangs suspended, to gain length, the thought being that with careful preservation of the splenic vessels at the fundic end of the stomach, satisfactory anastomoses could be accomplished in the neck, employing a branch of the external carotid artery and a jugular vein. In any case, preliminary trials with the method in the dog have been unsuccessful.

My first experiences with the use of the sternotomy incision, migrating the stomach into the thorax in this manner were in patients with large cancers of the lower esophagus and the gastric fundus, in whom the short residual antral fragment would not reach the divided esophagus. The problem resolved itself into finding a shorter path or swinging up a mobilized jejunal loop. And preservation of some fragment of the stomach, if it will suffice, every surgeon at such a moment in a difficult operation, feels instinctively constitutes the simplest and the happiest solution in the accomplishment of restoration of continuity of the tract.*

*In recent months, for high-lying intrathoracic cancers, the mid-line sternotomy incision has been extended only as high as the fourth interspace. Later, in the operation, a long incision is made in the second interspace on the anterior chest wall. When the stomach is severed from the esophagus, the stomach is migrated up into the thorax, through an aperture made in the right diaphragm just above the left lobe of the liver, in line with the liver-notch. The stomach is brought up anterior to the lung. Any excess in length of stomach is cut away. In patients under 50 years of age with an active gastric secretion, the stomach is not an ideal substitute for the esophagus. The only good thing to be said for it is its availability and ease of transfer. For cancer of the cervical esophagus, migration of the splenic end of the transverse colon into

Esophagitis

With a view to the avoidance of stricture formation, mucosal apposition by the open method of suture is preferable, when the stomach is to be united to the esophagus, as Churchill and Sweet[5] (1942) have pointed out. Yet, some of the narrowing occurring at such anastomotic sites may very well be due to the erosive



A B C

Fig. 1—X-ray films of Mrs. E. K. before and after gastric resection for "idiopathic" (acid-peptic) esophageal stricture.

A. Prior to operation April 1944. (A and B reproduced from *Surg., Gynec. & Obst., Vol. 88*).

B. Shortly after gastric resection (operation August 14, 1944).

C. Film made in October 1951 in California. Courtesy Dr. Stanley Moore. The contrast between A and C is startling.

action of acid-peptic juice upon the esophageal mucosa. For it has become increasingly evident in recent years that the esophageal mucosa is very vulnerable to injury by the gastric juice.

The experience of this observer has shown quite definitely that idiopathic strictures of the esophagus can be overcome by gastric resection in the same

---

the neck, with the cecum anastomosed to the gastric antrum, following excision of the entire acid secreting area, may prove to be the best procedure.

It is highly desirable in every esophageal cancer to excise a wide margin on the proximal side to insure a better chance of cure. That has been one of the shortcomings of esophageal resection in the hands of most surgeons. The approach described herein readily permits excision of a wide margin of normal esophagus proximal to the lesion. For cancers of the lower third of the esophagus division of the azygos vein with anastomosis at or above that level will lessen materially the frequency of incomplete excision of the cancer. In order to minimize undesirable side-effects of a very short residual esophagus, I have come to employ two expedients in the early convalescent period: 1. maintenance of the inlying intratracheal tube *in situ* until the patient is fully awake, thereby lessening the hazards of intratracheal aspiration of mouth, esophageal and gastric secretions; 2. retrograde placement of a number 14 French 4-hole duodenal tube just beyond the duodenojejunal ligament — this tube to lie in the stomach, with suction applied to it, well into the recovery period, thereby minimizing the hazard of reflux of bile and gastric juice into the trachea.

manner that, gastric resection deals effectively with a duodenal ulcer. The first of these patients, Mr. L. W., University Hospital No. 643715, dealt with in this manner was operated upon by the writer eleven years ago. He had undergone more than 100 esophageal dilatations for stricture in the preceding five years. After gastric resection (9/2/39), esophageal dilatation became unnecessary[6]. He can now swallow any food without difficulty. Mrs. E. K., University Hospital No. 672670, had a cork-screw like stricture of the thoracic esophagus (Fig. 1). She too had undergone more than 100 dilatations of the esophagus. By the time that Mrs. E. K. appeared on the scene, I had done two gastric resections for idiopathic stricture, with excellent functional results, in both. In both these instances, however, the relief of the stricture was an unanticipated but a very acceptable and gratifying result of operations undertaken primarily for the relief of peptic ulcer. In Mrs. E. K.'s case, there was no known or demonstrable peptic ulcer. However, Mrs. E. K. did give a history of hematemesis, and by 1944, I had come to the belief, which I still hold, that, hematemesis signifies an acid-peptic linked source of the bleeding. In the instance of the first two patients with stricture, no special authorization for performance of gastric resection was necessary. Both patients had conventional indications. In Mrs. E. K. that evidence was lacking. With no support and some opposition from the internists as well as from members of my own Staff, but with the approval of the patient after I informed her of the relief of the stricture experienced by the first two patients – and with some reservations over the likely success of the undertaking, a gastric resection was done. Two dilatations were necessary in the convalescent period, but no additional dilatations have been necessary during the period of seven years which has elapsed since then. A recent film is shown in Fig. 1c. Mrs. E. K.'s interesting story follows:

Case history of Mrs. E. K., 58 years of age at the time of admission to the University Hospital (U.H. #672670) in 1944.

This patient had come intermittently to the University Hospital since October, 1938. She then gave a story of dysphagia of 3 months' duration. In January, 1936, the patient had a massive hematemesis followed by melena. An abdominal exploration was done elsewhere shortly after an episode of hematemesis, at which time a large mass was found in the upper abdomen, and a diagnosis of polycystic disease of the liver and pancreas was established. Subsequent to the operation two subsequent episodes of hematemesis were experienced by the patient. Large abdominal masses were observed here on physical examination; moreover, the patient was found to be anemic, the hemoglobin being only 63 per cent. The white blood cell count was normal with a normal differential. Red blood cells were 2,890,000. Plasma proteins, blood cholesterol and nonprotein blood nitrogen values were normal when the patient was first seen here. Liver function tests were within normal limits.

X-ray examinations demonstrated a stricture in the lower 10 centimeters of the esophagus. The suggestion of a congenitally short esophagus was made.

Esophagoscopy revealed a stricture 31 centimeters from the incisor teeth. In the x-ray film the stricture appeared to cover a distance of 3 to 4 centimeters. A pebble-like appearance caused by the inflammation of the dilated portion of the esophagus was reported.

Because of the stricture, dilatations were instituted beginning with a No. 31 French bougie; the first dilatations were carried up to a No. 39 French. The patient was dismissed from the medical service with a diagnosis of esophageal stricture, "probably on the basis of a ruptured esophageal varix with secondary scarification of the esophagus".

Over a period of the next few years the patient was admitted frequently to the medical service of the University Hospital here for dysphagia, hematemesis, or other circumstances requiring hospitalization. On one hospital admission a cystocele was repaired; the patient intermittently had swelling of the ankles; there was some hypertension with electrocardiographic evidence of myocardial damage, probably the result of an earlier myocardial infarction. The blood pressure varied between 150 and 185 systolic, with diastolic recordings in the area of 90 to 100. Passive congestion of the lungs and liver was diagnosed. The patient also had emphysema. The plasma proteins were quite consistently under 6 grams per cent, and on one occasion as low as 5 grams per cent.

Gastric analysis was made on several occasions, and free acid without histamine was 28 degrees with a total acid of 54 degrees; on another occasion with histamine the free acid was 34 degrees with a total of 50 degrees. However, the recovery in quantity of acid from the stomach was unusually large on each occasion. The patient had occult blood in the stool quite consistently, and the hemoglobin varied between 7 and 11 grams.

Despite monthly dilatation, the patient's nutrition seemed to worsen, and because of the difficulty in swallowing and the fairly persistent anemia, and repeated episodes of bleeding, the patient was referred for excision of the stricture in the esophagus in April, 1944. The patient lived in the northern section of the state, almost 400 miles from Minneapolis and had been contemplating moving her home to this area to lessen the difficulty of travel made necessary by the frequent dilatations. The stricture began apparently in the upper portion of the lower third of the esophagus, and because of the patient's poor condition, operation at that time was not recommended.

The majority opinion both in the medical and surgical staffs favored esophageal resection. However, resection of the mid-esophagus was then essentially an untried operation even for esophageal cancer. I advised the patient that I had done gastric resection for 2 patients with "idiopathic stricture" of the esophagus with complete relief from need for dilatation of the esophagus. The patient accepted the suggestion of gastric resection which was done August 14, 1944.

At operation it was found that the patient had a congenitally short esophagus with a small paraesophageal hernia. A left subcostal incision was used. At operation a fairly large cystic liver was found, the left lobe being as large as a normal sized right lobe. There were several large cysts, some of which held more than 3 ounces of fluid. Many adenomas were noted in the liver. A few small cysts were felt along the left kidney and there were many cysts in the pancreas. The right kidney seemed small and it was not certain whether or not it contained any cysts. Adhesions were noted from the omentum down to the left lower quadrant. Many diverticula were felt in the descending and pelvic colon.

It was impossible to pass a duodenal tube through the stricture into the stomach prior to operation. The first step in the operation was to make a gastrostomy opening, through which a catheter was passed up through the esophageal stricture. Passage of the catheter was rather difficult; a small No. 22 bougie was finally pushed through. The catheter was caught above and sutured in place and a duodenal tube was pulled down. Then we used a larger bougie. Finally a fairly large bougie went through the stricture. The duodenal tube was left in the stomach, the gastrostomy opening was closed and the resection was carried out. A 75 per cent resection was done, together with excision of the pylorus and antrum. A Hofmeister retrocolic type of gastrojejunal anastomosis was done with two rows of sutures, the anastomosis being made at the ligament of Treitz. The duodenum was closed with 2 rows of fine interrupted silk sutures over Petz clips. The catheter in the stomach was pushed into the proximal duodenojejunal loop. The adhesions to the left lower quadrant were all divided. There was also an area in the small intestine, perhaps in the mid-ileum, where the gut was badly kinked by adhesions. These were cut and the bowel was made free. A tear in the surface of the bowel necessitated resuturing with fine interrupted silk. Interrupted silk sutures were used for closure of the abdominal wall. The patient stood this rather long operative ordeal quite satisfactorily and left the operating table in good condition. Despite this fact, convalescence was rather slow, and the patient stayed in the hospital until the first of September, at which time she was discharged. On September 12, she appeared in the Out-patient Clinic and said that she could swallow without difficulty and was eating bread and meat. At this time she returned to her home in International Falls and was not seen in the Out-patient Clinic again until December 13, 1944. At this time a No. 43 French bougie went down without difficulty. The plasma proteins on December 24, 1944, were 7.1 grams per cent. She appeared to be improving continuously despite the circumstance that her appetite was none too good. On February 21, 1945, a dilator was again passed, and it was found that a No. 45 French bougie went down readily. After this there appeared to be no further need for dilatations. In February, 1946, a sound was passed and was found to go down without difficulty. In August, 1946, the patient was hospitalized for a cold and because of asthmatic bronchitis. The presence of arteriosclerotic heart disease and polycystic liver and pancreas was again reaffirmed on physical examination.

There has been no recurrence of hematemesis and no difficulty in swallowing since the time of the operation in August, 1944. The patient's hemoglobin has continued between 12.5 to 13 grams per cent since operation. She eats everything and seems to be getting on quite satisfactorily and has gained considerable weight. An x-ray examination of the esophagus made in September, 1944, a month after the operation, showed some narrowing of the distal third of the esophagus. An x-ray examination made 2 years later indicated that the esophagus was normal. A recent film is shown in Fig. 1c. The patient has remained well and has had no difficulty in swallowing, nor bleeding.

Summary:—This patient had polycystic disease of the liver and pancreas, a congenitally short esophagus, and an esophageal stricture of unknown origin. There had been several episodes of hematemesis and it had been reasoned that bleeding was caused by esophageal varices followed by scar formation and esophageal stricture. Esophageal dilatation had been done approximately 100 times over a six year interval. Her nutrition was poor and edema and hypoproteinemia were present. Resection of the esophageal stricture had been urged but gastric resection was carried out on the thesis that the esophagal stricture was probably a manifestation of ulcer disease. The result justified the means. The patient has remained well and swallows without difficulty. The relief of the stricture by gastric resection is readily apparent in a glance at Figs. 1a, b and c.

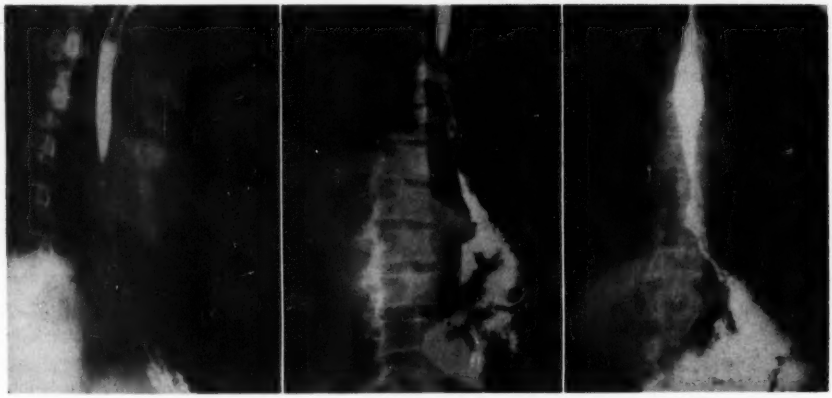
The case history of Mr. H. B. is equally interesting. Moreover, I have had so many inquiries addressed to me since the publication in 1949, that I welcome this opportunity to bring the case records of Mrs. E. K. and Mr. H. B. up-to-date, together with recent x-ray films showing the present situation in the esophagus of each patient. As the films (Figs. 2a, b and c) suggest and the clinical records attest to characterize the improvement as dramatic is in no sense an exaggeration.

When first seen in 1947, Mr. H. B., aged 47 years (U.H. ♯780565) gave a story of peptic ulcer since 1911. In 1940 he had a duodenal ulcer perforation which was closed surgically. Six weeks later the obstruction of the esophagus had progressed to the extent that he could not swallow his saliva and a gastrostomy was done to permit feeding him. This was done at the Wisconsin General Hospital. Since performance of his gastrostomy he has been able to swallow liquids only periodically, usually having to expectorate saliva as well. In the summer of 1947 his physician, Dr. William Focke, of Poynette, Wisconsin was in my Tuesday morning Outpatient Clinic. A Mr. C. G., (U.H. ♯772864) had just returned a few weeks after gastric resection performed for an extensive esophageal stricture, and reported himself as considerably improved. Dr. Focke said he had a patient who in many respects was very much like the case of C. G. and volunteered that he would like to send his patient here.

The patient arrived in July, 1947, and an effort was made to examine the esophagus with barium, but so little barium went through, that the examination was unsatisfactory. An attempt was made to use the esophagoscope and this also was unsatisfactory. Finally, a No. 5 ureteral catheter was passed down the esopha-

gus into the stomach and was fished out through the gastrostomy opening. A silk thread was then brought back and anchored to the cheek. After dilatation reexamination was done with barium, and it was found that a stricture extended down from the midsegment of the thoracic esophagus to the stomach. With many dilatations it became possible to dilate the esophagus up to about a No. 29 French. At that juncture, the patient felt much better, so he left and was not seen for several months.

Meanwhile, January, 1948, another perforation of a duodenal ulcer occurred. This time the patient was treated conservatively by suction applied to the gastrostomy tube and he appeared to get on satisfactorily. When he reported here for observation again in April, 1948, the esophagus again appeared completely occluded. The esophagus was gradually stretched up to a No. 29 French again. The



A B C

Fig. 2–X-ray films of Mr. H. B. before and after gastric resection for "idiopathic" (acid-peptic) stricture of esophagus.

A. Prior to operation, June 4, 1947. (A and B reproduced from *Surg., Gynec. & Obst., Vol. 88*).

B. Shortly after gastric resection (operation March 19, 1948).

C. Recent film (September 1951). Contrast the remarkable improvement over situation shown in A.

patient's hemoglobin at this time was 14.3 grams; leucocytes numbered 10,500. There was a large volume of acid in every sample aspirated. Volumes of 45 to 50 cubic centimeters were present in each specimen, including the fasting specimen; the free hydrochloric acid fasting was 42 degrees and after histamine rose to 74 degrees. When the patient came here he was thin, very weak, and it was difficult for him to stand.

At the time of operation on March 10 a granuloma containing purulent material was found in the retrogastric space suggesting that the perforation in January had occurred into the lesser sac. There was a hard, thick fibrous plastic area plas-

tered up against the transverse colon, which lent the impression in the preoperative x-ray film of a stricture there. The stomach was large. It became necessary to make an open closure of the duodenum. This was made with a single row of interrupted silk sutures. The duodenojejunal ligament was dissected down with some difficulty and 80 per cent of the stomach was resected.

The removed gastric segment, devoid of omentum, weighed 227 grams. A single row gastrojejunal anastomosis was made and the inlying duodenal tube was placed in the proximal duodenojejunal loop. Because of the presence of the subclinical retrogastric abscess encountered (absence of fever and leucocytosis), air vent suction drains were put into the subhepatic area on the right and behind the spleen on the left side.

The patient convalesced satisfactorily, but was kept in the hospital for a period of two weeks after operation to encourage better feeding. About the time the patient left the hospital it was found that a No. 29 French bougie was inserted without any difficulty. Two weeks later he returned and dilatation was accomplished up to a No. 33 French without any difficulty. A few days later it was found that a No. 35 French bougie could be passed without much difficulty; at this juncture, the patient returned home. He returned for observation again May 5, approximately 6 weeks after the operation, and said that he was eating well and had no difficulty in swallowing. At this time the esophagus was dilated to a No. 37 French with little difficulty. May 18, the patient returned at our request and it was found that a No. 37 French bougie went through without great difficulty. The patient is having very little difficulty in swallowing. Moreover, the x-ray film made at that time (May 18, 1949) showed considerable improvement in the lower reaches of the stricture but still showed some evidence of spasm or narrowing at the upper end of the esophageal stricture. The patient continued to improve and found that he could eat meat, hard rolls, celery, and carrots without difficulty. Prior to the operation, it is to be remembered the patient had difficulty in swallowing his saliva.

Summary:—The preceding case history documents the story of a patient of 47 years with a history of periodic severe difficulty from a duodenal ulcer since the age of 10. Eight years prior to admission here, surgical closure of a duodenal perforation was done followed a few months later by gastrostomy for feeding, because of the patient's inability to swallow his own saliva, occasioned by the stricture of the lower half of the esophagus. The improvement following a gastric resection has been dramatic. Today, this patient can eat any food without difficulty. He has full-time employment and has been completely rehabilitated. No esophageal dilatations have been done in the past 3 years. On April 25, 1950, a number 42 French Bougie was passed without difficulty. The last film made September 4, 1951 shows the present situation in the esophagus (Fig. 2c).*

*In the earlier publication [Surg. Gynec. & Obst. **88**:560 (May), 1949], the suggestion was made that, gastric resection might benefit a patient with a stricture owing to ingestion of

"Spontaneous" (Acid-Peptic) Perforation of the Esophagus

In his Balfour Lecture at Toronto, twenty years ago, the late distinguished Harvey Cushing[7] entitled his address: "Peptic Ulcer and the Interbrain". One of his illustrations showed a long linear slit in the esophagus of a patient who had undergone an operation for brain tumor. The circumstance of "spontaneous" perforation of the esophagus has been observed by other neurosurgeons. Up until recently, it has not been well understood. Accumulating evidence, however, suggests rather definitely that, it is owing primarily to regurgitation of the acid-peptic juice of the stomach into the esophagus. Regurgitant vomiting is not so infrequent an occurrence in patients with increased intracranial pressure.

"Spontaneous" perforation of the esophagus is not a primary hydrostatic phenomenon, for it takes more than an atmosphere of pressure to rupture the normal esophagus in the dog. On the contrary, the stomach will rupture with employment of an intraluminal pressure one tenth as great. The average value for the point of rupture of the esophagus in a small group of dogs was 869 mmHg.; for the stomach it was 86 mmHg.

That "spontaneous" perforation of the esophagus is owing to regurgitation of acid-peptic juice into its lumen is attested by the following circumstances: 1. when the pylorus of dogs is obstructed and the animals are given 30 mg. of histamine-in-beeswax intramuscularly each day for 4 days, (parenteral fluids being given meanwhile) perforation will occur in 50 per cent of the animals; 100 per cent of the dogs will exhibit evidences of severe esophagitis, owing to the regurgitation of the acid-peptic juice; 2. whereas severe esophagitis will attend the dripping of N/10 hydrochloric acid into the lumen of the esophagus at a fluid pressure of 20 cm., perforation of the esophagus does not follow. However, when gastric juice of the same pH, obtained from an isolated canine fundic pouch is dripped into the esophagus, perforation occurs quite regularly. In fact, a cat's

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lye. That suggestion was followed by a deluge of letters from many quarters of the globe asking whether it had been done. One needs frequently to be able to persuade other colleagues in his own area of the validity of such unconventional suggestions. The first opportunity to do this came in a patient, Mr. F. O. aged 47, University Hospital #828741 in December 1951. He had swallowed a strong mineral acid in November 1951. Excision of the entire acid-secreting area of the stomach was carried out, leaving the entire esophageal stricture which reached to the level of the tracheal bifurcation. Following this procedure the patient has been much better. He is still in need of esophageal dilatation, however. At present, a number 39 French bougie passes without difficulty. He can eat most foods without difficulty.

Two additional patients with similar strictures extending the entire length of the esophagus resulting from the ingestion of caustic agents have been treated by esophagogastric anastomosis; one, the anastomosis was made in the neck; in the other, at the apex of the thoracic cavity. Both patients are well and are not in need of dilatation. For emotionally unstable patients, this procedure is probably preferable to the long drawn-out therapy still necessary following gastric resection in the patient with a long, caustic stricture.

Professor Charles Auguste of Lille, France has informed me recently (March 17, 1952) that he has carried out gastric resection for lye stricture with an excellent result.

esophagus may rupture 20 minutes after the dripping of acid-peptic juice at a pressure of 20 cm. into its lumen.

These occurrences suggest definitely that, "spontaneous" perforation of the esophagus is owing to the regurgitation of acid-peptic juice into its lumen. Moreover, it is high time in the light of the experimental demonstration of these occurrences to suggest deletion of the prefix "spontaneous"; the correct designation, obviously, should be acid-peptic ulceration and perforation of the esophagus owing to regurgitation of gastric juice.

A fuller account of the experiences of this laboratory in the production of perforation of the esophagus was made by Ferguson[8] et al about a year ago. Cross and this writer[9] were able to show too that, erosion of the esophagus in the dog could be produced by bile and pancreatic juice. No injury of the esophagus was observed from *succus entericus*. These observations attest to the great sensitivity of the esophagus to chemical injury by the digestive juices. They lend increased credence to the belief that, idiopathic strictures of the esophagus in man are, in all likelihood, a manifestation of acid-peptic disease of the esophagus. The observance of esophagitis in the dog following the introduction of bile and pancreatic juice into the esophagus has suggested that, when total gastrectomy is done in man for cancer, restoration of continuity should be achieved by employment of the Roux Y principle of operation. In practice, this operation has already proved to be superior to the conventional terminolateral, end-to-side anastomosis between the esophagus and the jejunum, even when an enteroanastomosis is added to the later procedure.

Since Barrett[10] (1946) reported the first successful surgical attack upon "spontaneous" perforation of the esophagus, a large number of successes have been reported. It is a diagnosis which this writer once suggested could be made over the telephone. The important thing is to close the perforation without delay.

Megaesophagus or Esophageal Dystonia

Recently, I have dealt with this subject at length elsewhere[11]. Hence, I shall not say much about it here. This entity has essentially two components: hypertonus of the distal segment, from which occurrence it has derived the name cardiospasm, first applied to the disease by Mikulicz. There is, however, another element in megaesophagus which is at least as important, and that is, atony of the more proximal reaches of the esophagus. And herein lies the explanation of the huge bag-like, tortuosity of the esophagus which one sees not infrequently in this condition. Great dilatation of the esophagus will be seen only in megaesophagus or probably better called esophageal dystonia. The esophagus dilates because it has lost its tone. A bag-like esophagus is synonymous therefore with esophageal dystonia. In stricture and cancer of the esophagus, one sees only mild dilatation of the esophagus. In the presence of the normal tone of the esophageal wall, only

slight dilatation of the esophagus can occur as is apparent in the patients with esophageal stricture whose films are shown herein.

Esophageal dystonia is in many respects like Hirschsprung's disease of the colon. Absence of the ganglion cells of Auerbach's parasympathetic nerve plexus is, apparently, the underlying cause of both conditions. In esophageal dystonia absence or a paucity of these cells occurs throughout the atonic area. In man, it is to be remembered the distal two-thirds of the esophagus has smooth, involuntary muscle, whereas the proximal one-third has striated muscle. Esophageal dystonia concerns only the unstriated distal two-thirds of the esophagus.

The treatment of esophageal dystonia is hydrostatic dilatation – a bloodless rupture of the circular muscle fibres of the hypertonic distal narrow esophagus being desired. Periodic dilatation with bougies obviously, is not curative. If one or two attempts at cure by hydrostatic dilatation fails to cure, operation is in order. The experimental evidence cited above, outlining the great sensitivity of the esophagus to injury by acid-peptic juice suggests the reason for failure of the Heyrovsky-Gröndahl operation in which an anastomosis is made between the dilated esophagus and the gastric fundus. Similarly, clinical evidence indicates that the Wendel operation of open cardioplasty, in which a longitudinal incision is made through the narrow, hypertonic area, followed by transverse closure also invites esophagitis. The extramucosal myotomy of Heller apparently is a far better operation on this score in that, it does away with the hypertonus of the distal segment and is not followed by esophagitis. Persistent dilatation of the atonic proximal reaches of the esophagus usually persists, however. This writer has proposed straightening of the tortuous, dilated esophagus, excising as much of it as can readily be reached through an extrapleural sternotomy incision, together with excision of the entire acid secreting area of the stomach. A concomitant extramucosal Ramstedt myotomy also is performed on the pyloric sphincter to insure satisfactory emptying. This procedure appears to be a very satisfactory operation for the bag-like atonic megaesophagus of esophageal dystonia. Seven patients have been operated upon in this manner with satisfying results.

Paraesophageal Hernia

Because of the great difficulty of obtaining satisfactory exposure, most surgeons have abandoned the abdominal approach in the repair of paraesophageal hernia in favor of the thoracic. Merendino, Varco and the writer[12] (1949) suggested migrating the esophagus out into the leaf of the left diaphragm as a method of securing a satisfactory closure of the diaphragm about the esophagus. That it is virtually impossible to effect a satisfactory coaptation of the diaphragmatic crura from the thoracic approach, every surgeon has learned to know. Hence the origin of the suggestion of moving the esophagus out into the substance of the left diaphragm.

The sternotomy incision has changed all this (Fig. 3). It is now possible by doing a median sternotomy, cutting the sternocostal juncture out extrapleurally into the fourth, left interspace to get ready access to the lower reaches of the esophagus and the esophageal crura. This incision, accompanied by an upper mid-line abdominal incision going to the umbilicus has made repair of a paraesophageal hernia an operation as simple in most instances as the repair of an inguinal hernia.

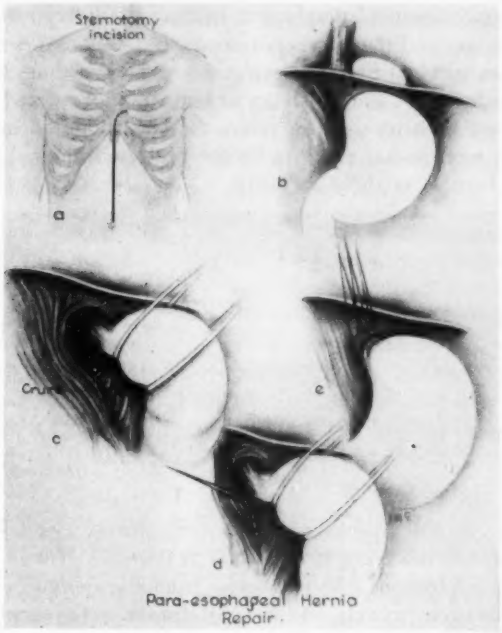

Fig. 3—Repair of paraesophageal hernia.

A. The extrapleural sternotomy incision for surgery of the upper abdomen and lower esophagus. For operations concerned primarily with the stomach, the sternum is split into the fourth left interspace. For megaesophagus, in which exposure of the stomach and the lower third of the mediastinal esophagus is desired, a T bar into the fourth interspace on the right as well as the left side is in order. By cutting down upon the diaphragm in the midline anteriorly until the pericardium comes into view, the job of the house-officer who pulls on the retractor is made lighter.

B. The hiatal hernial orifice containing the gastric fundus.

C. The stomach retracted with two tapes, uncovering the diaphragmatic crura.

D. Placement of a few 00 silk sutures in the crura.

E. Anchorage of esophagus to margin of diaphragm with several 00000 silk sutures.

Technic:— The subdiaphragmatic esophagus and the fundic end of the stomach are elevated with broad tapes (Fig. 3). The edges of the crura beneath the esophagus are carefully identified. The greater curvature of the gastric fundus can be detached from the spleen or it can be removed. The upper peritoneal at-

tachment of the stomach to the left leaf of the diaphragm is severed. The contents of the hiatal orifice come readily, ordinarily, into the abdomen. Employing four or five sutures of No. 00 silk, the crura below the subdiaphragmatic esophagus can be coaptated snugly. An additional eight to ten sutures of 00000 silk are employed to suture the edges of the diaphragm to the esophagus around the entire perimeter of that viscus. The first of these two maneuvers constructs a firm ring of support about the esophagus; the latter effects a snug-fitting, collar-like apposition between esophagus and diaphragm, making the interposition of tissue between the diaphragm and the esophagus impossible (Fig. 3). A sufficiently large number of paraesophageal hernia repair have now been done through this approach, with results which are satisfying to both the patient and the surgeon, to suggest definitely that sternotomy simplifies the operation and insures a satisfactory repair. This method has come to be the preferred method of dealing with paraesophageal hernias in this Clinic.

In a few patients with a concomitant gastric ulcer, a simultaneous segmental gastric resection has been done. In a few patients in whom there has been no demonstrable ulcer at the time of operation, but in whom the presence of an ulcer diathesis is suggested by repeated hematemesis, anemia, and by the presence of high acid values in the gastric secretion, I have done a bilateral subdiaphragmatic vagotomy, together with an extramucosal myotomy upon the pylorus. In the presence of an active duodenal ulcer with scarring, the pyloric myotomy has been of the Heinecke-Mikulicz variety. These additional procedures in patients whose stories suggest the presence of the ulcer diathesis have been distinctly worthwhile.

The left crus of the diaphragm is invariably stronger and heavier than the right. As one inspects the crura at operation, it becomes evident that, as the left crus shortens in respiration, it moves away from the esophagus. The powerful contraction of this strong muscle, and its separation from the esophagus in respiration – these two factors are probably the responsible initiating causes of paraesophageal hernia.

Esophageal Varices

In the Lister Lecture[13] (1945) it was pointed out that, the bleeding of portal hypertension was in all likelihood an acid-peptic linked phenomenon. The great sensitivity of the esophagus to chemical injury by the digestive juices was still unknown. It was recognized however that, excision of gastric tissue in excess of that necessary to cure the conventional ulcer diathesis was necessary It was soon learned in the experimental laboratory however, that portal hypertension constituted a severe abetor of the ulcer diathesis. And clinical experience too began to point out that, when protection against injury of the esophageal mucosa by gastric juice is desired, *all* the acid secreting area of the stomach must be excised.

In the first patient Mr. F. K., aged 59 (U.H. #750272), operated upon for bleeding esophageal varices on the basis of portal hypertension, who also had a cancer of the right lung, a 90 per cent gastric resection together with splenectomy was done (2/23/45), the right lung being subsequently (4/4/45) excised. Prior to operation, the patient was given a liter of blood a day for ten days because of repeated hematemesis. Esophageal varices had been ligated through a thoracic approach elsewhere a few months previously. That procedure had failed to obviate the episodes of hemorrhage which necessitated three hospitalizations prior to the gastrectomy. The portal pressure at the time of gastric resection was 25 cm. of saline solution. Ascites was present. In September, 7 months after the operation, blood was noted in the stool for the first time since the gastric resection. Anasarca became progressive and when the patient died 12/11/45, signs of liver failure were well developed. At autopsy, thrombosis of the portal vein and cirrhosis of the liver and esophageal varices were demonstrated.

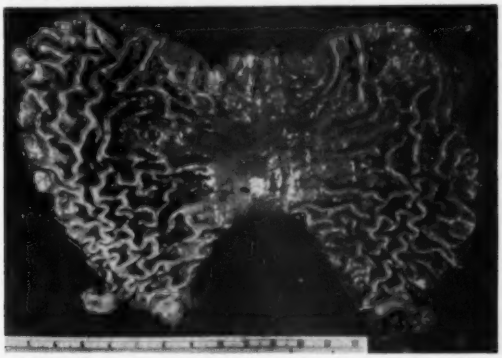

Fig. 4–Bleeding esophageal varices in a boy of 6 years (K. S.) who underwent excision of the entire acid secreting area of the stomach because of repeated episodes of hematemesis. A. The extent of excision of stomach and esophagus.

Subsequently, I extended the excision to include, in succeeding patients, as much as 95 to 98 per cent of the stomach. In 1947 Phemister and Humphreys[14] reported upon 2 patients with bleeding from esophageal varices; in one, an esophagofundic excision was done; in the other a total gastrectomy. In the meanwhile however, portacaval venous shunts had come into being, an operation which justifiably provoked greater interest amongst surgeons than gastric resection for esophageal varices, for it approached the problem more directly. Yet, there is one factor inherent in the problem which that operation deals with only indirectly, insofar as a portacaval venous shunt is successful in reducing portal pressure: bleeding from esophageal varices had been looked upon primarily as a hydrostatic problem; this writer on the contrary believed and had stated that, the acid-peptic linked factor was an item of great importance in the esophagogastric bleeding of portal hypertension[13, 15]. The experimental evidence cited above under the caption of esophagitis has supported that thesis. It was observed also[16] that abet-

ment of the ulcer diathesis in dogs followed employment of vasoconstricting agents as well as vasodilators (nitroglycerin) a circumstance which suggests that hemostasis in the mucosa exposed to gastric juice is an important determinant in the occurrence of peptic ulcer.

By 1948, the experimental work on esophagitis outlined briefly above was well on its way and presently, it became quite evident that the entire acid secreting area of the stomach should be excised – an operation proposed in the Alvarenza Lecture[17] of 1949 for both megaesophagus and for bleeding esophageal varices, an anastomosis between the esophagus and the antrum being advised, together with an extramucosal pyloric myotomy. This operation, it would appear, is a very satisfactory operation for the bleeding of esophageal varices. Moreover, it takes into account the circumstance that, the bleeding is probably not primarily hydrostatic in nature, other than insofar as an increased portal pressure abets the ulcer diathesis. Arroyave, Clatworthy and the writer[18] (1951) observed that an area of gastric mucosa the size of a penny migrated up into the esophageal wall on an adequate vascular pedicle – that such a small island of gastric mucosa implanted in the esophageal wall would invariably cause an esophageal ulcer, even when the remainder of the stomach was excised. Of all the mucosal surfaces exposed to injury by acid-peptic juice, the esophageal is by far the most sensitive. And, if our stomachs emptied via the esophagus rather than the duodenum, we would all probably have erosive esophagitis.

The typical situation in my experience in the bleeding of esophageal varices is shown in Fig. 3. The sternotomy incision permits excision of the lower 4 to 6 cm. of the esophagus, and when accompanied by a complete excision of the acid-peptic secreting area of the stomach – such an operation provides protection against the bleeding of esophageal varices. Moreover, this operation is more acceptable to the patient too than is total gastrectomy. Bleeding has not been observed in patients after operation in whom either complete excision of the acid secreting area or total gastrectomy has been done. This circumstance too lends firm support to the writer's thesis that, the acid peptic factor is an important item in the bleeding of portal hypertension. Linton[19] (1951) employs a thoracic approach in the acute emergency of bleeding from esophageal varices and opens the lower esophagus, suturing over the area of bleeding. Employment of the sternotomy incision, in suitable patients, who could tolerate the more aggressive procedure, would probably permit more effectual treatment in that, the lower bleeding area of the esophagus could be excised together with the entire acid secreting area of the stomach.

It is perhaps not out of place to record, herein, the case record of a boy with bleeding esophageal varices from portal hypertension for whom a total gastrectomy was done as an emergency operation after the transfusion of large quantities of blood.

Mr. C. G., (U.H. #764840), age 16 was first admitted to the medical service on 4/23/46 because of repeated episodes of hematemesis. There had been many hospitalization elsewhere for hemorrhage and anemia. A previous splenectomy had been done 7 years ago. The patient had been hospitalized several times on the medical service. On one of these admissions, the patient was seen by me and total gastrectomy was recommended. This suggestion was not accepted by the Medical Service, it being felt that the only surgical procedure which would be of any help to the patient would be an Eck fistula performed for the portal hypertension, which was undoubtedly present.

With periodic transfusions and continued observation the patient did fairly well. In June, 1947, the patient was readmitted on the Medical Service. The hemoglobin was 4 grams. A number of transfusions were given. On some days as much as 4 liters or more of blood was given. After the episode of hemorrhage was over, the patient seemed to do fairly well without any transfusions. Then early in July while still under continued observation on the Medical Service, the patient began bleeding again, and on July 8th after the transfusion of 4,500 c.c. of blood,

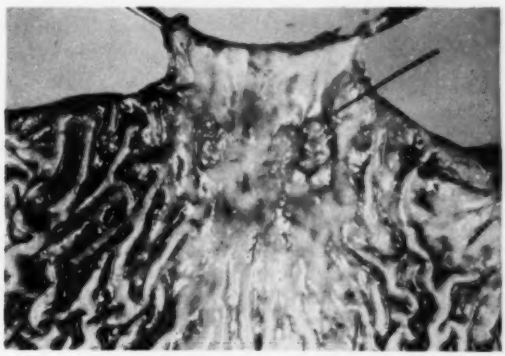

Fig. 4B–The varix (see dark pointer).

an emergency total gastrectomy was done. A check of the records suggest that in the area of 65 transfusions were given this patient for hemorrhage, over a relatively short interval of time. And it should be said to the great credit of the Blood Bank and the Clinical Laboratory that, no reactions occurred nor was the patient jaundiced at any time. The excised stomach weighed 295 grams. The operation was done without event and the patient convalesced nicely. The liver appeared normal and biopsy was normal.

He has been observed many times in the Out-patient Clinic since the operation. There have been no recurrent episodes of hemorrhage. The patient's hemoglobin has been maintained at a normal value. There has been no ascites. However, there have been during the past year a few episodes of jaundice. Peritoneoscopy and functional hepatic studies suggest the presence of nodular cirrhosis.

Since total gastrectomy was done, the patient has completed his undergraduate college training and is now a student in the College of Dentistry. In the main, his general health is good. Obviously, the prognosis must be guarded because of the presence of cirrhosis.

The following case record details the story of a young boy who underwent excision of the entire acid secreting area of the stomach together with 5 cm. of the esophagus for bleeding esophageal varices. This is probably the best direct manner in which to combat such hemorrhage, for experience has shown that the bleeding varix is often located, as in this instance (Fig. 5), right at the esophagogastric juncture.

Mr. K. S., age 6, (U.H. #819215). This patient was first seen at the University Hospital on the 24th of May 1950 because of hematemesis. There had been infrequent vomiting of large quantities of blood, followed by bloody stools. The hemoglobin had been as low as 5 grams. The blood picture otherwise was essentially normal. Several transfusions were given prior to splenectomy to bring the hemoglobin to a satisfactory level prior to operation.

The spleen was excised on 6/23/50 by Dr. David State. The excised spleen weighed 290 grams and was called splenic fibrosis or Banti's syndrome by the pathologist. The histological venture of a bit of liver excised was said to be normal.

The patient got along quite satisfactorily after the splenectomy with a gradual return of the hemoglobin value to normal.

In June, 1951, there was recurrence of bleeding with severe hematemesis. The patient was brought into the hospital with a view to having either a portacaval shunt or excision of the acid secreting area of the stomach done. The latter operation was decided upon, and was carried out through an extrapleural sternal splitting incision.

Complete excision of the acid secreting area with 5 cm. of the esophagus was done on the 29th of June 1951. An extramucosal myotomy was done also on the pylorus. The patient convalesced satisfactorily. There has been no bleeding since the operation. The patient was seen in the Out-patient Clinic in August, and again in October 1951 and is in the best of health. The patient was in the Out-patient Clinic again on the 29th of January, 1952. His parents reported that he has done very well. He is eating well and is gaining weight. There has been no suggestion of hemorrhage and the hemoglobin values are within the normal range.

Some Reflections on Surgical Management of Peptic Ulcer

And finally, in concluding, it probably would be proper to add a few remarks concerning the surgical management of peptic ulcer. The evidence from the laboratory and the clinic, cited herein, has taught the important lesson that, of all the mucosae exposed to acid-peptic juice, the esophageal is the most sensi-

tive and susceptible to injury. A part of the explanation resides, no doubt, in the meager capacity of esophageal mucus secreting glands, to store, as well as to secrete mucus, as contrasted with those same glands in the stomach and in the duodenum. Additionally, there is probably a greater inherent susceptibility of squamous epithelium to injury as contrasted with glandular epithelium. The observations cited herein, especially with reference to megaesophagus and esophageal varices suggest that, when the physiologic cardioesophageal sphincter is to be sacrificed, the entire acid-secreting area of the stomach must be excised, or erosion and ulceration with hemorrhage and esophageal narrowing are certain to follow. Vagotomy fails to add protection against this occurrence as is suggested in the experiments cited above in which an island of gastric mucosa was transplanted to the esophagus. Moreover, clinical observation too supports this experimental observation.

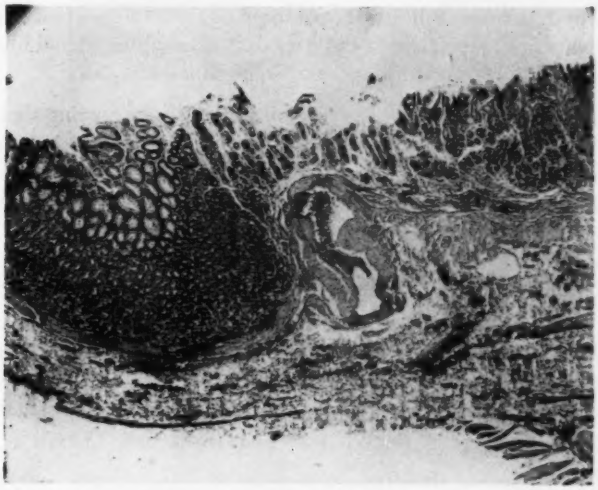

Fig. 4C—Ulceration of mucosa over the esophageal margin of the esophagogastric mucosal juncture (X100).

During the past ten years, a problem being worked upon in this clinic, more or less continuously, has been the surgical management of peptic ulcer. There is no ideal operation for peptic ulcer. It is regrettable that, an operation as simple as vagotomy should have proved an inadequate operation for peptic ulcer — a conclusion which appears to be well supported by the accumulated experience from surgeons everywhere.

Of the resection technics, the Billroth I and II operations, when accompanied by excision of 75 per cent of the stomach appear to be satisfactory operations in the surgical management of peptic ulcer. In the Billroth II operation, two other items appear important: 1. the antral and pyloric mucosa must be excised com-

pletely; 2. a short afferent duodenojejunal loop must be employed in the anastomosis. If these two conditions are not met, the risk of stomal ulcer is increased greatly.

Elsewhere[20], the writer has summarized an experience with segmental resection in which the antrum has been preserved in continuity with a tiny residual fundic pouch. That experience suggests definitely that, the gastrin secreting mucosa of the antrum may be retained, if it is not separated from the residual gastric pouch. In fact, this is what reflection suggests is the weakness of the Eiselsberg, as well as the Devine, pyloric exclusion operation. No recurrent ulcers have been observed in 80 patients submitted to segmental resection. I had hoped that the "dumping-syndrome" could be avoided completely in segmental resection for peptic ulcer. That, unfortunately, has not been an important gain in the procedure – though, in the main, those symptoms, together with weight loss, have been less prominent than in the Billroth II operation. Segmental resection has important technical advantages, especially in the very difficult supraduodenal ulcer crater – a condition commonly alluded to as "inoperable duodenal ulcer".

The greatest gain, however, has probably been in reorienting surgical reconsideration of the role of the antrum in the surgical management of peptic ulcer. Despite the circumstance that, the prophecy of failure[21] has been forecast for segmental resection, the fact remains that, this operation does thwart the ulcer diathesis. Moreover, the experimental evidence from the laboratory, employing the histamine-in-beeswax method of studying the promise of any proposed operation for peptic ulcer also supports the thesis that, segmental resection is an acceptable operation for ulcer[22].

All this has occasioned the writer to hark back to an earlier experience with tubular resection and gastrojejunostomy[23], a modification of Connell's fundusectomy[24], a proposal which the writer made and carried out more than ten years ago.*

It would appear that the stomach is essentially two organs – the acid-peptic secreting digestive organ, and the non acid, gastrin secreting area of the antrum. The testimony of the experience of surgeons with the Eiselsberg antral exclusion

*Since this presentation was made, I have resurrected the operation of tubular gastric resection for duodenal ulcer, closing the residual gastric pouch transversely. That arrangement makes for a more capacious gastric pouch. In patients with a deformed duodenum, a pyloroplasty of the Heinecke-Mikulicz variety has been added. If the pyloric sphincter can be retained intact, the "dumping-syndrome" may be considerably less in evidence than following the conventional technics of gastric resection. In reverting to this type of procedure, already one gain has been noted: inasmuch as the antrum, the strongly motile segment of the stomach retains its vagal innervation in this procedure, the depression of appetite, which is commonly observed in the early weeks of convalescence following segmental resection, as well as after the more usual types of gastric resection – this unwelcome complication does not appear to be so definitely in evidence. Pursuit of this inquiry, both in the laboratory and in the clinic, promises to be interesting, and, fruitful. Of the various resection technics which have been carried out in the clinic, this operation appears to be the most promising.

operation and the protection afforded against recurrent ulcer in segmental resection suggest, as indicated above, that it is *separation* of these two organs which spells the doom of leaving antral mucosa in the Billroth II procedure. This is an area, for thought and practice, in which the surgeon must secure better orientation than he has now. It is a problem which will receive careful study in our surgical experimental laboratory during the coming year.

Summary

A number of surgical conditions of the esophagus have been discussed briefly. The surgery of esophageal cancer has made important strides in the last dec-

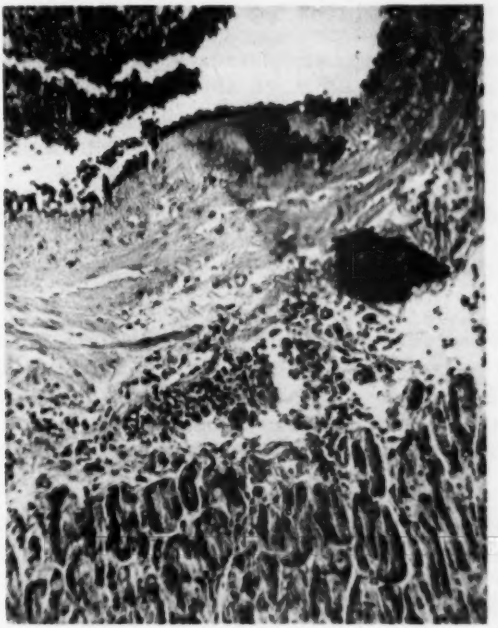

Fig. 4D–The same area under higher magnification (X250). Loss of blood by hemorrhage is understandable.

ade. The great importance of Ohsawa's contribution (1933) to the surgery of esophageal cancer merits special mention.

Idiopathic stricture of the esophagus is essentially acid-peptic disease of the esophagus and a manifestation of the acid-peptic ulcer diathesis. A three-quarter gastric resection, as carried out for duodenal ulcer, corrects the stricture. A follow-up on 2 patients with high grade esophageal obstruction, who underwent gastric resection in 1944 and 1948 respectively is reported herein. The premise

that idiopathic esophageal stricture is acid-peptic stricture of the esophagus receives strong confirmation in the remarkable relief observed in these striking cases following gastric resection.

The designation "spontaneous perforation" of the esophagus should be replaced with acid-peptic perforation of the esophagus, for the ease and regularity with which perforation of the esophagus may be brought about by perfusion of the esophageal lumen with gastric juice suggests definitely that the reflux of gastric juice is the cause of "spontaneous" esophageal perforation.

Megaesophagus is essentially esophageal dystonia owing to hypertonus of the distal segment and atony of the dilated portion. When hydrostatic dilatation fails to relieve the situation, operation is in order. For lesser grades of esophageal dilatation, the extramucosal cardiomyotomy of Heller is usually effective. For the baglike, tortuous atonic esophagus, straightening of the esophagus together with the excision of the entire acid secreting area of the stomach is in order. With sacrifice of lesser lengths of the acid secreting area, esophagitis and hemorrhage are bound to follow, because of the great sensitiveness of the esophagus to chemical injury by gastric juice.

The sternotomy incision lends itself particularly well to repair of paraesophageal hernia, making an otherwise difficult procedure a relatively simple one. The technic of the hernial repair with employment of the sternotomy incision is illustrated.

The role of the gastric juice in the genesis of the bleeding of esophageal varices accompanying portal hypertension is stressed once more. Excision of 4 to 5 cm. of the lower esophagus, together with the entire acid secreting area of the stomach, apparently, affords satisfactory protection against recurrent hemorrhage. The typical finding has been bleeding from an erosion at the esophagogastric juncture.

A brief discussion has been made of the various surgical resection technics for peptic ulcer. It is pointed out that it is the *separation* of antrum from the rest of the stomach that constitutes the hazard of retention of the gastrin secreting area in the Billroth II type of gastric resection. This situation exists also in the antral exclusion operation of Devine. The success of the segmental resection in thwarting the ulcer diathesis indicates definitely that, when the antrum remains *attached* to the small residual fundic pouch, no hazard of ulcer recurrence obtains. Moreover, more recent experience suggests that retention of vagus innervation to the antrum and pylorus is probably a desirable feature of a satisfactory operation for peptic ulcer. Tubular gastric resection accompanied by transverse gastroplasty is an operation which meets satisfactorily the requirements of an acceptable and adequate operation for peptic ulcer.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS*

S. A. MACKLER, M.D.

Chicago, Ill.

This presentation was primarily a motion-picture, preceded by a discussion as outlined.

1. The physiologic mechanism whereby rupture of the esophagus occurs is described.

2. The historical aspect of the disease is briefly noted.

3. A case is presented, and the significant diagnostic findings are described. Preoperative radiographic films are demonstrated.

4. The operative treatment is shown. The motion picture film shows the thoracic approach to the rent in the esophagus. Gastric content is seen lying free in the pleural cavity and upon the intrathoracic viscera. The manner in which it escapes from the team in the esophagus is also seen. The method of surgical repair is demonstrated.

5. Postoperative radiographic films are demonstrated.

Discussion

Dr. O. H. Wangensteen:—I should like to reecho what Dr. Lermann has said about this presentation. It was really very nice seeing the movies and hearing Dr. Mackler talk about this condition.

I have not seen many patients with this entity and it is only recently that surgeons have been successful in treating it; however, during the wee hours of one morning approximately twenty years ago, I did diagnose the occurrence of "spontaneous" perforation of the esophagus in a patient over the telephone. It happened very much like this: My resident, Dr. Herbert A. Carlson, called me up to say he had a patient in the hospital on whom he was prepared to operate for a perforated ulcer; however, the patient had some emphysema in the neck. He had been in the outpatient clinic that day, and a patch of leukoplakia in the mouth, had been fulgurated under local anesthesia.

I asked Dr. Carlson, "Has the patient fits of coughing?"

"No."

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*Abstract of a paper presented before the Course in Postgraduate Gastroenterology of the National Gastroenterological Association, Chicago, Ill., 20, 21, 22 September 1951.

"Well, he must have a perforation somewhere in the esophagus. Give him some sterile barium to verify this."

That was done. Well, we did not invoke any treatment other than to put in an indwelling duodenal tube; and that, obviously is not enough. A perforation of the stomach may close with suction and even so may a perforated duodenal ulcer. In the esophagus, however, a long, linear slit, such as Dr. Mackler showed us, there is little likelihood that such a perforation can close without surgical intervention.

I was very much surprised to see the extent of the pleuritis that developed in seven hours in Dr. Mackler's case, and I would think probably that, enzymes *per se* may do as much harm as the bacteria. We surgeons should lend some thought to this occurrence in peritonitis.

We have good antibacterial agents. We need some studies on antienzyme action. Kunitz, working with Northrop on pepsin, has lately come out, as many of you know, with a soy bean trypsin inhibitor. This agent should probably be evaluated in acute pancreatic necrosis to note whether it exerts a protective action. We need a good antipepsin, too. Such an agent, if effective, might simplify the management of peptic ulcer considerably.

There is one point upon which I would take issue with Dr. Mackler. He stressed the hydrostatic factor in "spontaneous perforation" of the esophagus. As you know, stress on any point of a distended hollow viscus is the product of the internal diameter times the tension and Pi. Now, if, therefore, tension was the primary factor, you would expect the stomach to rupture and not the esophagus, for as you may remember, yesterday I indicated that the normal esophagus will not rupture under less than an atmospheric of pressure. The diameter of the esophagus is small, and its wall is thick. Rupture by hydrostatic rupture of the cecum from cancer of the distal colon is common. Every surgeon knows this. It takes three atmospheres of pressure to perforate the normal appendix. The lumen of a normal appendix holds only 0.5 c.c. at 40 cm. of saline solution pressure. Yet, after some hours of sustained intraluminal pressure, because of the devastating effects of pressure upon the circulation of the appendix, softening and rupture at a relatively low intraluminal pressure will occur. It came as a great surprise to Drs. Buirge, Dennis and myself that, the appendix secretes fluid. Every human appendix secretes 1 to 3 c.c. of fluid a day. Adolph Hitler talked a lot about Lebensraum and expansion. And, as you know, if 1 to 3 c.c. tries to occupy the space for which nature has made provision for only 0.5 c.c., and the exit is occluded so the fluid does not get out, a struggle ensues: the wall softens, the intraluminal pressure increases, and soon the wall rupture. When surgeons operate to remove a gangrenous appendix, they should remember that a normal appendix, which will not perforate at less than 3 atmospheres of pressure will rupture readily, when tugged at ever so slightly.

What cells in the appendix are responsible for this behavior? It would be good to know. The time must come when there will be in every medical school histochemists, like Dr. Gomori, working in the interphases of activity – the no man's land between the physiologist and the anatomist.

To get back to the esophagus, "spontaneous perforation" should be called acid-peptic ulcer and perforation of the esophagus. It is not hydrostatic in any sense, and the pepsin has much to do with it. A number of years ago, Lester Dragstedt proposed that peptic be deleted in the designation; that the proper appelation is *acid* ulcer. The esophagus and its sensitivity to chemical injury is a proper testing ground for that thesis. When N/10 hydrochloric acid is dripped into the cat's esophagus, esophagitis follows but perforation is difficult to produce; when, on the contrary, gastric juice obtained from an isolated canine gastric pouch is used perforation may occur within twenty minutes after the dripping of such juice is commenced – that sensitive is the esophagus to injury by acid-peptic juice!

We are just beginning to learn a few things about the susceptibility of the esophagus to injury by the digestive juices. Those of you who saw my colored lantern slides yesterday will recall that, bile and pancreatic juice together may produce esophagitis too – yes, even when the entire stomach has been excised. When the bile and pancreatic juice containing segment of the duodenum in dogs is attached to the lower esophagus in dogs, followed by excision of the stomach, esophageal erosion, with hemorrhage and anemia, occurs. If our stomachs emptied via the esophagus instead of the duodenum, we would all have esophagitis.

Harvey Cushing, in the Balfour Lecture in 1932 spoke of "Peptic Ulcers and the Interbrain". The reason that neurosurgeons see this entity, and they have observed it more often than other clinicians, is that, with increased intracranial pressure there may be considerable vomiting. And under that circumstance acid-peptic juice is refluxed into the esophagus; mucosal erosion, ulceration and perforation may occur in consequence.

In gun shot wounds of the neck, the appearance of emphysema should suggest the possibility of penetration of the esophagus. Here too as in acid-peptic perforation of the lower reaches of the esophagus, an x-ray film will detect the escape of air much more quickly than will the hand, by palpation. Bubbles of air can be seen in the tissues on an x-ray film before palpable crepitus can be detected.

Dr. I. Snapper:– I am very gratified to see that the methods which Dr. Boerhaave used around 1700 have been justified now by Dr. Mackler's experiences. Dr. Mackler may be interested to know that Professor Boerhaave was severely criticized for the methods he used and which enabled him, among others, to make the first observation of the remarkable syndrome discussed today. Boerhaave is often praised as the first teacher who took his students to the bedside. This, how-

ever, was done in Padua long before Boerhaave; one of the Hollanders, older than Boerhaave, brought this educational method from Italy to Holland. However, Boerhaave not only took his pupils to the bedside, but if he could obtain an autopsy, he took his pupils to the opening of the cadaver. Hereby he exposed himself to severe criticism. His older colleagues insisted that he emphasized too much the morbid processes that happened during the last days before death and thus taught a *pathologia cadaverosa*. It may well be true that this criticism was partly justified. Nevertheless, we see in 1951 that at least part of Boerhaave's observations and educational methods are correct.

Occasionally, an excessively dilated stomach may perforate. I have never seen, so far as I can remember, an esophagus perforation under these circumstances.

Dr. Walter Freyman (*West New York, N. J.*):– Were there histories of peptic ulcer of the esophagus or other ulcers in any of the cases operated for ruptured esophagus in your series? Secondly was there any symptom of peptic esophagitis in the cases, where rupture was caused by vomiting or other extraneous reasons?

Dr. Saul Mackler: I wish to thank the previous discussers for their comments.

In answer to several questions just voiced, there has been no history of preceding esophageal disease in these two cases or in the cases that have been presented in the literature. Esophageal rupture has occurred in the following circumstances – not only after vomiting but following straining at defecation; straining lifting a heavy weight after a heavy meal; during a convulsive seizure in an epileptic patient. In several instances of seasickness, persons with no previous alimentary disturbances ruptured their esophagi. It has also occurred following intracranial and other operative procedures, during postoperative vomiting and following anesthesias, and following obstruction of the lower intestinal canal.

I feel there is a differentiation between perforation and rupture. If an organ perforates due to previous disease, we expect to see a round or oval eroded opening, which we do see with *perforation* of the esophagus. With *rupture* of the esophagus there is no round or oval opening but rather it is a longitudinal split, as you have seen in the motion picture. I have taken fifty postmortem esophagi and subjected them to increased intraluminar pressure, the pressure being distributed throughout the entire esophagus. In all cases with one exception, the esophagus ruptured in one area and in a linear or longitudinal burst, as you have seen. The configuration is the same, the extent of the rupture is the same, the region is the same, when the rupture is thus produced experimentally as when found clinically.

There has been an accidental *in vivo* experiment in a human. In jest, a worker with a compressed air pump put the tube in his mouth, clamping the tube shut with his hands at the moment he had it in his mouth; however, he accidentally slipped and let go of the tube and the air rushed down his esophagus. He

died. At postmortem examination his esophagus was rupured. The pressure came the opposite way, but the esophagus presented the same logitudinal split at the lower end..

Furthermore, I have taken biopsies of the mucous membrane in these cases of spontaneous rupture of the esophagus. I don't have the pictures with me, but the specimens show only acute inflammatory changes, some congestion of the vessels. I justify this acute inflammatory response on the basis of the trauma and because the esophagus was exposed to gastric juices, as you saw in the film, following rupture. If there had been a preceding inflammatory reaction and ulceration prior to perforation, one would expect at the margin of the mucous membrane to see necrosis of tissue, or some sort of chronic inflammatory reaction. There was none. There was no necrosis of the edge of the mucous membrane, no necrotic tissue around it at all, as one would expect to see at the edge of a perforation.

Further, on postmortem microscopic examination in cases previously reported, they, too, found no necrosis or evidence of chronic inflammation, or ulceration, or erosion.

I am relatively alone, I suppose, in this, and perhaps it is presumptuous for me to continue to maintain that this is due to hydrostatic forces, in the face of the eminent persons who spoke before me, particularly Dr. Wangensteen; nevertheless, from what I have seen, I feel I must continue to maintain this stand.

HISTOCHEMISTRY OF THE GASTROINTESTINAL TRACT*

G. GOMORI, M.D.

Chicago, Ill.

Recent advances in histochemistry have made it possible to identify chemically and to localize accurately a number of substances in the gastrointestinal tract. In the past these substances had been known only by their morphological aspects or by chemical assay of relatively large samples of tissue or secretions. The data to be presented here are fragmentary because most of the methods are so new that they could not yet be applied systematically to all normal tissues, and even less to the study of pathologic conditions.

1. Mucin:—"Mucin" or "mucin-like substances" are terms which have slightly different meanings for the chemist and for the histologist. To the chemist mucin-like substances are a group of polysaccharides composed mainly of amino-sugar, various other sugars, uronic acids and, in some cases, sulfuric or phosphoric acids. To the histologist the definition of mucin is tinctorial rather than chemical, although two of the typical staining reactions of mucin are true histochemical tests based on the formation of aldehydes from polysaccharides by oxidation. These two reactions, the Bauer[1] and the McManus[2] stains, are positive with all known varieties of mucin, whereas other staining methods, empirical in nature, give variable results with different types. For instance, one kind of mucin, found mainly in the goblet cells of the small intestine and the colon, stains quite intensely with mucicarmine and mucihematein, and is metachromatic with toluidin blue. Another type, present in the surface and neck cells of the gastric fundus, remains unstained by mucicarmine and shows no metachromasia with toluidin blue; on the other hand, it may stain with Best's carmine[3]. The latter type of mucin is called "mucoid" by Schaffer[4] (caution: the term "mucoid" is used in entirely different and unrelated senses by Lim[5] and by Zimmerman[6]). Aldehyde fuchsin[7] differentiates between two types of mucin: one is stained very intensely and the other one remains almost completely unstained. The dividing line between these types of mucin is not a sharp one: in almost every slide a large number of cells showing intermediate and/or overlapping staining properties can be observed.

As mentioned, the Bauer and McManus stains have a definite chemical meaning while the interpretation of the other staining reactions is not clear. Lison[8] believes that true (i.e., alcohol-resistant) metachromasia with toluidin blue is an exclusive property of sulfated polysaccharides. However, this theory cannot be accepted any longer in face of recent observations. Clara[3], Francini[9]

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*Presented before the Course in Postgraduate Gastroenterology of the National Gastroenterological Association, Chicago, Ill., 20, 21, 22 September 1951.

From the Department of Medicine, The University of Chicago.

and Bignardi[10] have shown that treatment of the sections with chromic acid will abolish the metachromasia of mucin but make mucoid, in fact, even glycogen and starch, metachromatic. The explanation of the staining of mucin and mucoid by mucicarmine and Best's carmine, respectively, on the basis of "salt formation" (Clara[3]) is chemically unsound. The problem of the chemical differences between various types of mucin is a challenging one and would deserve thorough investigation.

2. *Other polysaccharides:*—Black-Schaffer[11] found that the foamy cells of the mucosa and of the lymph nodes in cases of Whipple's disease contain, besides fat, large amounts of a polysaccharide, intensely stained by the method of McManus. The chemical nature of this substance is not known except for the fact that it is not glycogen.

3. *The secretion of hydrochloric acid:*—According to the textbooks of histology, hydrochloric acid is considered to be secreted by the parietal cells. However, there is no direct evidence to support this thesis. Indicators or reagents for inorganic acids have been parenterally administered to animals while their stomachs were secreting. No signs of high acidity or alkalinity could be observed anywhere within the mucosa itself. The first traces of acid appeared on the free surface, within the lumen proper[12, 13]. This finding is suggestive of a mechanism by which no acid, but rather a non-acid substrate, is actually secreted. In the lumen this is then converted into free acid, probably by enzymatic action. This possibility has never been systematically investigated.

4. *The enterochromaffin substance:*—The enterochromaffin (argentaffin, "yellow") cells of the intestinal mucosa have been known for over 80 years[14], and their unusual staining (or rather chemical) properties have invited much speculation. There are indications that these cells may be concerned with the elaboration of an antianemic principle[15].

The most important chemical properties of the enterochromaffin granules are as follows:

a) They are soluble in alcohol and water; however, after fixation in formalin they are completely insoluble.

b) Dichromates stain them in an intensely brown shade[14].

c) They reduce alkaline silver solutions to metallic silver, without the addition of any extraneous reducing agent[16].

d) With diazonium salts they yield intensely colored azo-dyes[17].

Cordier and Lison[17, 18] on the basis of the last three reactions, drew the conclusion that the granules must contain a derivative of catechol, some compound chemically related to adrenalin. Their theory became universally accepted, and its correctness had not been questioned until recently. However, on a more careful

analysis of the chemical reactions involved, it became obvious that the catechol theory is untenable. It can be proven that the reactions of the enterochromaffin cells are due to a derivative of resorcinol[19], not of catechol. This is a rather interesting discovery, because resorcinol has never before been known to be a constituent of animal tissues. The exact nature of the compound responsible for the reactions and its biological significance have not been determined.

Jacobson[20], on the basis of spectroscopic studies, came to the conclusion that the fluorescence of the enterochromaffin cells is due to a pteridine, a close relative of folic acid, if not folic acid itself.

Carcinoid tumors contain the same phenolic substance as the enterochromaffin cells. The biochemical maturation of the tumor cells may vary considerably: some are loaded with typically staining granules, whereas others contain very few or none at all.

5. *Enzymes:*—a) Alkaline phosphatase. This enzyme is abundant on the surface of the villi of the small intestine in most species[21]. In some species the Golgi apparatus of the cells in the same area also contains large amounts of the enzyme[22]. Relatively well-differentiated tumors of the small intestine may show a picture very similar to that of the normal mucosa.

b) 5-nucleotidase[23]. This is a special alkaline phosphatase the substrates of which are 5-nucleotides, such as muscle adenylic acid and inosinic acid. In the intestinal tract it is localized mainly in the muscle layer, and the colon contains much more than the upper segments.

c) Acid phosphatase is present in practically all the cells of the gastrointestinal epithelium, with the exception of the parietal cells[24]. (This is contrary to the findings of Seligman and coworkers[25].) Carcinomas of the stomach may contain a high concentration of the enzyme. Assay of the gastric juice for acid phosphatase has been proposed as a diagnostic test for malignancy[26].

d) Esterases. The classification of esterases is a complex and controversial problem which cannot be discussed here in detail. For practical purposes, esterases will be divided into two groups: lipases which preferentially hydrolyze long-chained fatty acid esters of glycerol (fats) and esterases proper which attack mainly short-chained esters of simple alcohols and phenols.

The lipases themselves can further be subdivided into two types only one of which is capable of hydrolyzing all fats, saturated or unsaturated. In most species, this enzyme ("true lipase")[27] is present exclusively in the pancreas; in the mouse (and possibly in some other species), also in the stomach. Lipases of other organs (including the intestines) are entirely incapable of splitting unsaturated fats although they hydrolyze saturated fats quite readily. Since it is known that unsaturated fatty acids are essential in the nutrition of some species, it is conceivable that some of the peculiar symptoms of pancreatic deficiency (celiac disease, sprue) might be due to the nonutilization of unsaturated fats.

In the human gastric fundus lipase is present in the chief cells. In the antrum and the remainder of the intestinal tract, it is found in scattered single cells which are morphologically indistinguishable from their neighbors, and unrecognizable without the use of specific enzymatic stains. The esophagus is intensely positive in the embryo but shows only a patchy reaction in the adult. Squamous carcinomas of the esophagus often stain strongly for lipase[28], and this may be a diagnostic feature in cases of lymph node biopsy. All other squamous carcinomas (with the exception of bronchogenic tumors, which may also be active) are uniformly negative.

Very little is known about the distribution of simple esterases. In man, the localizations of lipase and esterase are quite similar except that esterase is more widely distributed than lipase. Large amounts of esterase are present in the macrophages of the colonic mucosa and in some nerve cells, neither of which contain any lipase. In some species, especially the rat, at least two different esterases, distinguishable from each other by their specificity towards various naphtholic substrates, are present. In the duodenum of the rat, an esterase which hydrolyzes esters of *a*-naphthol is found in the villi, but it is absent from Brunner's glands, A different esterase, specific for esters of naphthol AS (a trade name for β-naphthol substituted in position 3) shows an exactly opposite pattern of distribution. Both esterases are present also in the ganglion cells of Meissner's and Auerbach's plexuses and in the muscle layer.

e) Cholinesterase[29] is present in the ganglion cells and the muscle layer of most species and in the epithelium of the small intestine of some species. In addition, the enzyme is also found in the cores of intestinal villi where its localization appears to be in the walls of the lymphatics.

f) Phosphamidase. Small amounts of this enzyme are present in practically all tissues. Much higher concentrations are found in epithelial malignant tumors[30]. Carcinomas of the gastrointestinal tract usually give a strong reaction, the intensity of which parallels the degree of histologic malignancy. Polyps of the colon form a most interesting exception; they are all intensely positive, regardless of their histologic appearance. Even the smallest, most innocuous looking lesions will show up in a sharp contrast against the unstained background. One is tempted to say that this observation may be the biochemical counterpart of the statistically established fact that such polyps are premalignant by nature.

Unfortunately, no methods are available for the localization of proteolytic and glycolytic enzymes.

A reasonably thorough knowledge of the topographic distribution of enzymatic activities under various conditions, when achieved, will represent an important step towards a better understanding of the physiology of the gastrointestinal tract in health and disease.

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Discussion

Dr. I. Snapper:—We are truly privileged that Dr. Gomori has been willing to discuss his highly interesting investigations which open new fields not only for the physiology but also for the pathology of the gastrointestinal tract. Of course, in doing so he has demolished a few of the holy houses which we have cherished for many decades.

The first thing is that he finds hydrochloric acid only in the lumen of the stomach and on the surface of the cells. The clinicians by indirect reasoning have always believed that an active acid substance was excreted by the cells of the stomach. If a patient develops calcinosis – that is, deposition of calcium in tissues outside the bones – certain organs are affected more frequently than others, to wit: the lungs, kidneys, and stomach. Clinicians have stressed that in all these organs acid substances are excreted: in the kidneys, acid phosphate; in the lungs, acid CO_2; and in the stomach, hydrochloric acid. In these three acid producing organs, an alkaline reaction must at least temporarily be present when the acid

is excreted. Thus when excess calcium is present in the serum, calcium hydroxide will precipitate most easily in these three organs.

Is this classical conception still correct in the light of Dr. Gomori's results concerning the hydrochloride acid secretion in the stomach?

Of interest is also the fact that the remarkable cells, the chromargentaffin cells, do not contain catechol but resorcinol. Does this revive the old and already forgotten treatment of disease of the gastrointestinal tract with resorcinol?

Maybe Dr. Gomori will discuss more in detail the action of the true lipase of the pancreas upon saturated and unsaturated fatty acids. In sprue and in celiac disease, the pancreas is considered to be completely normal because all function tests of the pancreas in these two diseases have been within normal limits.

The demonstration of a special enzyme in carcinoma and in polyps, which splits phosphocreatine and phospho-arginine, may open up a new future for the enzymology of malignancy. If this would mean that creatine and/or arginine are of great importance for the growth of tumors or polyps, then perhaps a new approach to influence the growth of tumors may have been opened. If tumors are so dependent on arginine and creatine, then we could feed the organism certain substances which are in biological competition with arginine and creatine. This would be comparable to the action of sulfa drugs against bacteria which mainly is due to competition of the sulfa drugs with the chemically closely related para-aminobenzoic acid. The bacteria needs the latter substance which cannot enter the bacteria because sulfa drugs compete with it.

We then could feed our polyps of the intestine (which I am afraid most of us have) a substance which has a chemical structure related to that of creatine and arginine. When this is done, our polyps perhaps will not grow so much and may not give rise to carcinoma.

Therefore, Dr. Gomori's investigations, which to some of us may look somewhat abstruse and far-removed from the field of practical gastroenterology, nevertheless offer us new and original methods which will permit us to find new approaches to seemingly insoluble problems.

Dr. George Gomori:—As far as the acid-producing site of the calcinosis theory is concerned, I don't think the fact that no actual acid is secreted in the stomach is against it. Whether the substance is excreted as acid or not, makes very little difference as long as it must be a hydrochloric ester. The chloride must come from the blood, leaving bicarbonate behind. Probably the same kind of alkalinity would be produced whether acid or non-acid is excreted.

Regarding true lipase, nobody has performed function tests using pancreatic secretions. The fact is that in all disturbances of this nature, the low iodine number of the blood lipids indicates non-absorption of unsaturated fats.

PROCTOLOGIC PRINCIPLES

JULIUS GERENDASY, M.D., F.A.P.S.
Elizabeth, N. J.

It is the purpose of this article to present a number of principles or axioms which, it is hoped, will be found of service in the successful management of patients with disease of the colon and rectum. For this purpose, an extensive review of the proctologic literature was undertaken. From it, selected portions were condensed into comprehensive statements or concise definitions each of which contain either a general doctrine or a self-evident truth. It is suggested that the quotations with references should be consulted for further details. For practical purposes the material was classified as follows: I. Anatomic Principles. II. Diagnostic Principles. III. Clinical Principles. IV. Surgical Principles.

I. Anatomic Principles

Knowledge of the normal anatomy of the anal canal is necessary in order to have a criterion to follow in the successful surgical treatment of pathologic conditions in the diseased bowel. This requires an understanding of the anorectal musculature, the levator ani muscle and the various subdivisions. Of importance, are the trilaminar nature of the external sphincter muscle, the longitudinal muscle fibre with its musculofascial extensions and the composition of the anorectal muscle ring in the surgical treatment of fistulae, hemorrhoids, fissure-in-ano and contracted anus. These muscles act as guardians of continence[1,2,3,4]. The arterial, venous, lymphatic and nerve supply to this portion of the terminal bowel are are also important[2,5,6].

The anal intermuscular septum produced by the downward fibromuscular prolongation of the longitudinal muscle of the rectum is an important landmark in the anal canal. It is easily palpated as a depression or groove around the anal canal. Incising it divides the subcutaneous external sphincter in conditions of tight anus or chronic fissure-in-ano. The internal sphincter can be palpated at the junction of the anal canal and rectum as the rounded inner boundary of the intermuscular septum. If the sphincter muscle be cut, control is preserved as long as this ring of muscle is not divided[1,2].

The anatomical subdivision into anal canal and rectum is of clinical importance. The junction of the rectal mucous membrane with the anal skin is known as anorectal, dentate or pectinate line. A knowledge of the differences in the anatomic structure of the anal canal and rectum is essential in understanding the symptoms which result from pathological changes in these tissues. The anal canal is lined with squamous epithelium and has a rich supply of

sensory nerves. As a result lesions external to the pectinate line produce a variety of (pain) sensations. The rectal mucosa is not difficult to identify. It is innervated by the sympathetic nervous system and as a result only sensations of pressure or fullness may be experienced. Careful analysis of the subjective symptoms, therefore, will frequently indicate the tissues involved in disease[7].

The anal canal from the standpoint of disease processes and their surgical management is divided by the intermuscular septum into the upper one-third or pecten, and lower two-thirds or anoderm. The pecten, as distinguished from the pectinate line is of practical clinical and surgical importance in an understanding of the pathologic processes present in this portion of the anal canal. The following important structures are contained therein: The preformed anal ducts and intermuscular glands, the recesses of the anal crypts, the lymphatics and capillary network all of which are of importance in anorectal suppurative processes and as sites of focal infection and its complications, since from here the infection spreads to the various perineopelvic spaces[2].

The pecten also marks the anastomotic zone between the superior and inferior hemorrhoidal plexuses, the dividing line between the somatic and visceral vascular chains; also the change in the nerve supply from the cerebrospinal nerves of the anal canal to the sympathetic nerves of the rectum. It is a site of acquired and congenital stricture. Its lower margin, the anal intermuscular septum, marks the usual location of the anal crypt and the internal opening of anal fistulae and it is the usual level at which suppurative processes extend laterally to the ischiorectal fossa. Finally, it is the site of predilection for anal fibrosis or pectenosis the pathogenic factor, particularly in fissure. The pecten should not be confused with the pectinate line[2].

The genito-ano-rectal lymphatics are of clinical importance to the proctologist because of differences in the lymphatic drainage in the male and female. In the male the extension of the infection and malignant metastasis involves the inguinal nodes and in the female the cervix uteri, its adnexa and the rectum.

The whole problem of these intramural lymphatics require careful study[2,5,8]. For example, pyogenic and tuberculous abscesses appear in the perianal tissues without apparent origin in the anus or rectum. This suggests that the lymphatics act as carriers of infection from within the rectum [2,9,10]. The general acceptance of this fact following the development of such infection, *in some cases,* would obviate search for a nonexistent internal opening and eliminate unnecessary loss of time and effort during the surgical procedure in anorectal abscess and fistula.

The histopathology of anal crypts reveal that cryptitis, anal fistula, periproctitic abscess and other infections have their origin in the infected narrow simple tubular or complex branching ducts which extend from the mucosa of the anal crypts and not in the crypts of Morgagni as previously taught. The conclusion is that in order to eradicate the common anorectal lesions, excision or drainage of these preformed epithelial tubules is necessary[11,12].

Anatomically internal hemorrhoids never arise in or from the anal canal and no matter to what degree they may prolapse they are still internal hemorrhoids arising from the superior hemorrhoidal vein and their lower margin is always marked by the dentate line[37].

The following classification is of value in fistula surgery: A fistula with an internal opening in the anal canal should be known as an anal fistula or fistula-in-ano. When the internal opening is in the anorectal line it partakes both of the anal canal and rectum and should be known as an anorectal fistula. When the internal opening is in the rectum proper, then it should be known as a rectal fistula. A horseshoe fistula, perineal fistula, etc., are all varieties of one or the other of the foregoing. The so-called blind external and blind internal fistulae are not fistulae at all but sinuses[13].

The anatomic classification of fistulae based on the relationship of the fistula tract and its internal opening to the anorectal muscle ring is surgically important. As long as part of the ring is left intact incontinence is avoided. All the anal sphincter muscles *external* to this ring may be divided in any manner without harmful loss of control. The ring is a composite fibromuscular band composed of the upper portion of the internal sphincter, the longitudinal muscle, the puborectalis portion of the levator ani and the external sphincter ani profundus muscle. For the competent and successful surgical treatment of fistulae and other deep seated anorectal infections, as well as in hemorrhoidectomy, the composition of the anorectal ring musculature is important[1,2].

A knowledge of the presence, location and limitations of the more clearly defined perineopelvic spaces is essential in the surgical treatment of fistulae. Those which are clearly defined are as follows: (1) The perianal space below the subcutaneous external sphincter muscle; (2) the circumanal space between the external sphincter and the internal sphincter muscles; (3) the submucus space; (4) the ischiorectal spaces below the levator ani muscles; (5) posterior communicating space beneath the anal intermuscular septum; (6) supralevator space above the levator; (7) posterior levator space below the levator; (8) retrorectal space between the rectum anteriorly and the sacrum posteriorly[2,14].

An understanding of the anatomy of the rectosigmoid is necessary for a safe and proper method of sigmoidoscopy. An understanding of the differences in the curving axis of the sigmoid and the abnormal position of the sigmoidal loop either from adhesions, a short mesentery, or extrarectal disease, aid in avoiding the possibility of perforation of the bowel during instrumental examination. Recognition of ulcers, adenomata, and pathologic processes of the rectosigmoidal area is also facilitated by an understanding of the rectosigmoid anatomy[2,15].

II. Diagnostic Principles

In the diagnosis of disease of the terminal bowel the symptoms of the patient should be looked upon as indications of various types of pathological lesions.

An adequate history and a proper examination will help determine the diagnosis. In anal diseases symptoms are usually concerned with disturbances in sensation, while rectal diseases show principally variations of function. This is because of the differences in the blood supply, innervation and lymphatic drainage[7].

Digital rectal examination precedes and exceeds in importance all other methods of anorectal examination. It is the most informative and the most indispensable of all methods employed by proctologists to determine the presence of anorectal lesions including carcinoma. Digital examination of the anorectum should always be preceded by a note of warning or "tap on the door", thereby avoiding painful sphincter spasm. The patient should then strain as at stool in order to relax the sphincter muscle.

The presence of lower abdominal symptoms especially if referable to the rectum necessitates a careful rectopelvic examination. With the patient in the left lateral Sim's position a bimanual rectoabdominal examination will give valuable information in pelvic infections of the male and female organs, surgical affections of the lower abdomen and pelvis (acute appendicitis, intussusception, ectopic pregnancy) malignancies involving the pelvic organs. A valuable but much neglected diagnostic procedure is here recommended[16].

The clinical significance of the rectal shelf (Blumer) in the pouch of Douglas is important to proctologists since metastatic deposits from malignancy in the gastrointestinal tract may be palpated here through the rectum and when found is of diagnostic and prognostic importance. Routine digital examination of this area above the prostate should be performed in every case[17,18].

The internal opening of a fistulous tract may often be palpated and, in fact, digital palpation of a fistulous tract is often far more valuable as a diagnostic help than the passing of probes.

The probe is an instrument of diagnostic value. Skillful utilization of it will be found a frequent aid during fistula surgery. Careful and persistent search with the exploring probe, used gently, will reveal the direction of a fistulous tract. A probe passed through the main tract will find its way to the internal opening of the anal canal or the point of the probe can be felt in the submucosa and indicate the point of origin of the infection[1].

The sigmoidoscope is an instrument by means of which a direct view of the interior of the rectum and sigmoid may be obtained in order to diagnose and treat diseases of the anus, rectum and sigmoid[19,20]. In the technic of proctoscopy or sigmoidoscopic examination the cooperation of the patient is of utmost importance. The proctoscope should never be passed beyond the rectal ampulla without first withdrawing the obturator and viewing accurately the rectum and sigmoid as the scope is being introduced. If the bowel is not properly prepared it is better to make another attempt after proper preparation[21,22].

Examination with the sigmoidoscope should be routine in all preoperative proctologic cases; also in patients with persistent diarrhea or the passage of blood and mucus and middle-aged patients complaining of "piles" or constipation of recent origin; also in all patients with abdominal symptoms and with whatever disease elsewhere in the body where the diagnosis is obscure[19,20].

It should be the rule that an x-ray following barium enema should be preceded by a proctosigmoidoscopic examination. *To the proctologist the purpose of the roentgenologic examination is essentially that of revealing changes in the colon above the reach of the proctosigmoidoscope.*

Immediate recognition of a perforation of the sigmoid as the result of diagnostic and therapeutic manipulation is mandatory in order to save life. An immediate operation is indicated. Should the situation permit the sigmoidoscope should be allowed to remain at the point of penetration for the purpose of identification of the site of perforation[23,24,25].

The essential laboratory procedures of diagnostic value in proctologic disorders include the routine hematologic and urinary tests; examination of the stool for blood, pus, ova, and parasites; the Gram stain for the gonococcus; the Frei (lygranum) test for lymphopathia venereum and the microscopic examination of excised tissue of either benign or malignant growth including suspected lymph nodes wherever found; also a serum test for syphilis, hematological test for hemopathies and the sedimentation rate.

A patient with a history of bleeding from the rectum should be considered to have a malignant lesion of the gastrointestinal tract until proved otherwise[26]. As a general rule fresh blood passed by anus comes from the anal canal. Blood coating the fecal mass is caused usually by a lesion in the lower bowel. If the blood is intimately mixed with feces (tarry) the lesion is high up probably gastric or intestinal[27].

Fresh blood in the stool of infants and children should suggest the presence of polyps either in the rectum or higher up in the bowel. Usually these are found within reach of the nine inch sigmoidoscope[28]. The proctologic examination of infants and children is a simple procedure requiring no general anesthesia. A mild sedative prior to examination is all that is necessary. By placing a child in a jack-knife position over an inverted Hane's table it is more managable and better cooperation is obtained[29,30].

Two varieties of epithelial tumors (polyps) occur in the rectum. The "pure" villous papillomas and the "pure" adenoma (an intermediate group can also be distinguished histologically as to which are papillomas on the surface and adenomas at the center). The first two each have their own characteristic appearance and histologic structure. Papillomas, because they arise superficially from the surface epithelium, proliferate into a villous type of tumor with a long fragile stalk. When the epithelial cells situated in the depth of the mucous membrane begin to pro-

liferate they encroach on the tissues below the surface of the mucous membrane to form an adenoma. Clinically adenomas are more likely to undergo malignant changes than papillomas. The importance of the intermediate group lies in that it indicates the importance of examining all parts of a polyp to exclude the possibility of malignancy[31].

A long-standing benign anal lesion may develop into a malignant lesion. Routine biopsy is absolutely essential in the surgery of all presumably benign lesions. One-third of malignant lesions were discovered by Tucker and Helwig during histologic examination of clinically benign lesions. Rosser's theory that cancer may develop in long-standing benign anal lesions was substantiated in six of these cases[32-36].

Polyps of the colon and rectum will become malignant if they are not already so when they are first discovered. Briefly, polyps are assumed to be precancerous lesions and are treated as such[37,38].

The Guaiac test is the most suitable for office use in the determination of occult blood in the stool. A positive Guaiac reaction with a definite change to blue or dark green denotes organic cause of bleeding in a high proportion of cases[39].

Biopsy in proctology is usually confined to the following three anatomical subdivisions: (a) *Anal biopsy*, for chronic hypertrophies, particularly on the edges of long standing fissures, openings of chronic fistulae, particularly of the submucus variety and skin and scar tissue hypertrophies: (b) *rectal biopsy*, suspected tissue should be excised in toto when it is possible to bring it out into the anal canal, thus saving all portions of the specimen. Where this is impossible a very fine wire loop electric cutting current is applied in order not to coagulate the most important part of the tissue (base) and vitiate the proper histological examination; (c) *sigmoidal biopsy*, is done with essentially the same technic as described for the rectum, that is, with the fine wire electric snare. Experience is necessary when an electric snare is used above the peritoneal reflexion in order to avoid penetration with peritonitis as a complication. Biopsy of tumors definitely above the peritoneal reflexion where the procedure is difficult due to sharp recto-sigmoid angulation or adhesions, the abdominal route with opening of the bowel is the preferable method of approach[40].

Indications for histologic examination of biopsy specimens that can be seen through the sigmoidoscope are. (a) in ulcers of doubtful origin; (b) in adenomatous growth; (c) in tumors for which radical operation is proposed or radium treatment instituted. Sections are taken from the edge of the ulcer, the base of a polyp or the surface of a fungating growth[41].

The study of the microscopic structure of tissues removed at operation is essential to distinguish a benign from a malignant condition. Every resected specimen should receive a routine histologic examination[42]. In the determination

of malignancy in polypoid disease, histologically the entire polyp together with its base should be studied[37].

About 20 per cent of rectosigmoidal cancers are treated for hemorrhoids surgically or otherwise and accentuate the necessity for complete and comprehensive examination of the terminal bowel[43].

III. Clinical Principles

The primary factor in the production of diseases in the anorectal region is infection. This is often concealed in the same manner as it is in the tonsils or teeth and may become a point of focal infection with secondary disease elsewhere in the body as a result. The treatment is surgical in order to drain infected tissue spaces[44]. Infection in the teeth, tonsils, sinuses, gallbladder and intestines is responsible for many anorectal disease processes and should be eradicated[45].

Constipation is the most common etiologic factor in many cases of anorectal disease. This is due to the repeated mechanical, chemical, allergic and bacterial trauma of the constipated stool. Anorectal hygiene is a much neglected prophylactic measure. Normal bowel habits and proper cleansing of the anal area will prevent serious disorders common to the terminal bowel[46].

The signs and symptoms that accompany anorectal disorders may be grouped into sensory disturbances, abnormal secretions or discharges, disturbances of defecation and local anatomic changes. By asking specific and searching questions regarding each of these items, one may usually build up a clinical picture that aids greatly in making the diagnosis and this may be confirmed by making a proper local examination[47].

Reflex disturbances due to anal diseases are numerous and varied. The symptoms are due to reflex irritation of the sacral spinal nerves. The more common are those associated with digestive disturbances and those relating to the genitourinary organs; epigastric distress and so-called intestinal indigestion (flatulence) are among the most common symptoms; dysuria, in both sexes; also dysmenorrhea and urethral spasm are other reflex manifestations. When the ordinary medications have failed to relieve the symptoms produced, a careful examination of the anal canal should be resorted to and if any disease conditions are found they should be properly treated[48].

Proctologic disorders are frequently intimately related to the function of the pelvic organs through the intimate correlation of the neural supply to these organs. The importance of anorectal diseases as a focus of infection and as a causative factor in malfunction in distant organs should indicate examination of the terminal bowel routinely and the location and eradication of the etiologic focus.

Pruritus ani may be caused by infected crypts, proctitis, constipation, cancer, fungus infection, clothing, alcohol, condiments, allergy to foods and any number

of other etiologic factors. The offender most frequently overlooked is Oxyuris vermicularis or pin worms.

The pain caused by a sharp foreign body (toothpick, chicken bone) in the rectum comes on suddenly during defecation. There is constant pain or discomfort in the rectum and sometimes also in adjacent parts, particularly on activity. The site of penetration is within the last inch or three-fourths inch of the rectum. When an abscess follows it begins to form within two or three days of the puncture. When the internal opening of the abscess can be identified it should be included (excised) in the wide drainage of the abscess[49].

IV. Surgical Principles

Only in a very few cases of extreme emergency should one operate without a complete examination resulting if possible in a definite diagnosis. A mutilating operation for syphilis, tuberculosis, dysentery or condylomata is a sad commentary on the surgeon's early training. It is not a rare occurrence for a busy careless surgeon to operate in the presence of a dormant ulcerative colitis or amputate for prolapse in paresis or operate upon ulcerated hemorrhoids in cases of malignancy[50].

In the consideration of the preoperative preparation of the proctologic patient there are two main points to be stressed; exclusion of associated diseased processes and second, the postoperative comfort of the patient. This latter concerns elimination of catharsis or soapy enemas. Laxatives prescribed preoperatively are conducive of gas pains, postoperative distention, adynamic ileus, excessive flatus (with the possibility of anal wound separation) and the disturbances in normal physiological activity of the colon as influenced by the activity of the stomach and therefore difficulty with bowel movement postoperatively[51].

It is underestimating proctology as a specialty to consider most operations too trivial for the use of a general (intravenous, low spinal or sacral) anesthesia. The patient is thereby surrounded with all the precautions necessary to the performance of adequate surgery and the prevention of sepsis and other complications. *One should not believe in "little" operations around the anorectum because it is fallacious to consider any surgical operation "little"*[52].

The basic concept in proctologic surgery is that the same diagnostic care, adherence to anatomic zones and gentleness in handling tissue are indicated in the anorectum as the surgeon employs in other areas of the body[53].

Chronic anorectal lesions (disease processes) are usually the result of repeated infiection. The inflammatory process has altered the anatomic structures to such a degree as to interfere with the function of the sphincter mechanism. Surgery attempts to return the parts to their normal anatomic relationship by the removal of gross interference with physiologic function.

The conservation of normal tissue is the first law of proctologic surgery. Preoperative estimation of the surgery necessary in each case should be routine. The surgical removal of normal anatomical structures will not improve physiologic function. It is only by removing diseased tissue which interferes with normal function that surgery is beneficial.

The anatomy of the terminal bowel is often distorted by disease processes and warrants careful reappraisal of the normal relationship of the parts to each other. Failure to do so will result in scar formation, contracted anal canal and susceptibility to recurrent infection.

In proctologic surgery it is no less important to know *what* to do than *how* to do it.

In planning the surgical approach it is important not to disturb, e.g. by divulsion or instrumentation, the gross picture of the existing anatomical deformity of the anal canal in order to avoid confusion between the normal and the diseased tissues, thereby less of the normal tissue will be sacrificed and better functional result will be obtained.

Removing too much of the papillary (dentate area) causes loss of the defecatory reflex sensation. This is especially apt to occur after hemorrhoidectomy and plastic operations, as for instance, rectal prolapse or anal stenosis[54].

The pecten area in the posterior anal canal is most vulnerable to fibrosis postoperatively. Therefore, operative procedures in this area should be minimal and postoperative treatment, including gentle digital dilation, should endeavor to avoid the formation of scar tissue as this interferes with the normal distensibility of the anal canal, especially during defecation[55].

Hemorrhoidal disease is caused by infection and the reason surgery is necessary is to drain the infection in it and in the deeper tissues. The injection treatment does not drain the infection. If anything, it seals it within the tissues[56, 57].

Surgical principles in hemorrhoidal diseases:—(a) Surgical excision is the indicated treatment for hemorrhoids and Salmon's ligature method is the safest and best operation. (b) The insertion or termination of the longitudinal muscle at the mucocutaneous line has a direct bearing on the pathology and surgery of the anal canal. When the firm subjacent attachment is lost by inflammation internal hemorrhoids prolapse subcutaneously producing large external-internal internal hemorrhoids. However, internal hemorrhoids are always covered by mucous membrane and external hemorrhoids are always covered by skin. (c) It is important that the internal pile pedicle to be ligated should be as narrow as is consistent with the inclusion of all the large vessels including the hemorrhoidal artery in the ligature. (d) If many piles are ligatured and their bases are left large and broad, when tied up they draw the mucous membrane together and cause a great narrowing of the anal canal. When the base of the pedicle is narrow

there is less pain; also the danger of postoperative hemorrhage is much less. (e) Stenosis (contraction) of the anal canal occurs in cases where the piles are ligated at the same level or where several piles are included in the ligature[57].

The technic which is important in a hemorrhoidectomy requires careful dissection which frees the external portion of the hemorrhoid and associated skin tags up to the subcutaneous external sphincter muscle in order to conserve the fibro-elastic extensions below the anal intermuscular septum. The strangulating ligature will then include the hemorrhoidal artery and avoid the possibility of secondary hemorrhage. When the excision is carried above the anal intermuscular septum it disengages the hemorrhoid, permitting it to retract into the rectum which is followed by a large raw area predisposing to postoperative hemorrhage, stricture formation and delayed healing. The stumps of the three primary hemorrhoids should lie no higher than the inner margin of the subcutaneous external sphincter muscle[1,2].

Severe hemorrhage following the operative treatment of internal hemorrhoids is often caused by: (1) postoperative infection; (2) trauma, especially from hard constipated stool; (3) the giving of sutures; (4) the retraction of the wound; (5) the failure to include hemorrhoidal artery in the ligature; (6) the action of the sphincter ani and/or sloughing of the wound cause (3) and (4); (7) other factors are anemia and general debility, and avitaminosis, blood dyscrasia, (hemophilia, leukemia)[58,59].

Pain following hemorrhoid operation is due to: (1) The anal skin is included in the ligature; (2) the fibres of the sphincter muscle are included in the ligature; (3) when the submucus tissue is adherent to the sphincter muscles postoperatively; (4) insufficiently large drainage wounds with lymphedema due to localized inflammation forming painful tags[58].

It is best to allow an excess of redundant anal skin following the operative procedure in order to avoid the possibility of anal stenosis. Usually such a redundancy will flatten out and if not it can be easily removed under local anesthesia as a minor office procedure. Superabundant skin which remains apparent after hemorrhoidectomy may be cut off at the time of operation but this should not be too freely excised for fear of contraction (anal stenosis) when the wound heals[58].

It should be the rule to use transfixion catgut ligature for the control of bleeding above the anorectal line; and for bleeding below this line use simple plain catgut ligature tie. Transfixion suture ligatures are dangerous when used in connection with infected wounds. Contrary-wise the less suturing, the less possibility of postoperative infection and scar formation.

All tissue surrounded by a surgical ligature is destroyed by sloughing and dead tissue is conducive to infection and slow healing. No one should fear infection where wounds are left open and sutures are absent.

The arterial supply to the hemorrhoid has not been sufficiently stressed as a factor in postoperative hemorrhage after hemorrhoid operation especially the insurance of proper and complete ligation of it[60].

When the dentate (mucocutaneous) line is not readily identifiable due to distortion by disease, the hypertrophied anal papillae may indicate the upper limit of the anal canal or in other words, of the external sphincter muscle which may, therefore, be cut without loss of sphincter control.

A knowledge of the presence, location and limitations of every potential perineopelvic space is essential in the surgical treatment of anorectal abscesses. All abscesses in these areas should be drained externally and never into the rectum[2, 4, 14, 61*].

Principles of fistula surgery:—Operative interference is so often ineffectual because (1) first and foremost the topography of the fistula is not thoroughly recognized and thus a part of the fistula tract escapes division. (2) Failure to follow the internal opening. (3) Failure as to proper after-treatment. (4) Operative timidity in estimating the amount of sphincter muscle that can be safely cut or the amount of muscle to be left and which can be relied upon to function successfully and maintain continence[62].

Whatever the nature of the fistula, the cardinal fact of fistula surgery is the location of the internal opening and its relationship to the anal musculature and the anorectal ring. These should always be palpable and recognized during the operative procedure. Every internal opening must be layed open before an anorectal fistula may be cured[63]

Horseshoe fistula, both anterior and posterior are treated by opening or excising the lateral tracts, packing the space and then excising the main tract leading to the rectum at a later date[62].

The healing of wounds is closely related to the efficacy with which lymphatics drain the part. In the presence of lymphedema with the consequence of poor drainage of the wound, the disposition of fibrous scar tissue is greater, also the susceptibility to local infection is greater[64].

In proctology it is important to bring one's dexterity and experience into play during the surgical procedure, but if anything, it is more important to give serious attention to the after-treatment if a cure is to be effected[65]. The patient should be visited and the operative field inspected daily while confined to the hospital. He should be kept under close observation until complete recovery satisfactory to the patient and surgeon is assured.

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*The drainage of a submucus abscess is the only exception to the rule.

The daily alaxative passage of a well formed stool postoperatively will massage the anal canal and avoid bridging and pocketing in anorectal wounds. The use of laxatives should be condemned.

The inherent resistance of the anal tissues to injury and infection and the ability to heal should be recognized in any endeavor to hasten the healing of postsurgical wounds.

To promote healing of wounds, surgical excision of diseased tissue is always necessary; an open flat wound thus obtained will heal by second intention. Healing by second intention, provided the surgery is correct maintains its own course in time with nature's rate and method of healing in spite of the application of lotions, ointment, soothing stimulant, antiseptic or caustic medications. Healing occurs by the growth of the granulation tissue at the base of the wound and by the covering of this tissue surface with epithelial cells from the skin or mucous membrane at the periphery. This method of healing is nearly always employed in rectal surgery and it is the only method in this area upon which reliance can be placed. The prevention of bridging, pocketing of overhanging skin, especially at the anal verge and of sinus formation is all that is necessary from the surgeon. Healing by second intention is then the rule as opposed to adherence of the walls of the wound, that is, the attempt to heal by third intention[1].

Surgical principles in wound management involve chiefly factors which aid wound healing. These may be summarized as (a) *Local factors*: (1) Careful handling of tissues during operative procedures in order to prevent excessive or unnecessary damage to normal tissues; (2) the avoidance of mass ligatures about the wound thereby insuring the integrity of the blood and lymphatic supply to the part; and (3) eliminating large or prolonged gauze drains in postoperative wounds since they interfere with drainage, act as foreign bodies and are conducive to infection of the wound. Other factors of prime importance are the prevention of local tissue infections by: Sharp dissection with a knife since it is least damaging to living tissues and provides the optimum wound for healing; adequate drainage of wounds about the anorectum in order to prevent local vascular stasis and lymphedema; excision of all diseased tissues to insure a healthy wound; complete hemostasis since blood is a good culture medium and, also, hemorrhage in a wound separates living tissue and increases the amount of work living cells must do to bridge the defect; and the use of minimal sutures, ligatures and ties as they act as foreign bodies and are conducive to infection. (b) *Systemic factors* that improve wound healing concern adequate protein nutrition to improve tissue repair; therapeutic doses of the water-soluble vitamins, especially A, D, and C and the treatment of secondary anemia where present[66].

The pre- and postoperative use of antibiotics and sulfonamides will be followed by the smaller incidence of abdominal pain and distentions, less tendency to fibrosis as the result of inflammation of tissues and will hasten healing[54]. In certain cases, however, sulfonamides which sterilize the intestinal contents in-

crease the tendency to postoperative bleeding. This is due to a prolongation of the prothrombin time and inactivation of Vitamin K.

Urinary retention after proctologic surgery is often caused by the packing inserted into the anal canal. It is also a frequent cause of postoperative pain. The insertion of any type of anal packing is contrary to the accepted principles of proper wound drainage in proctologic surgery.

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TREATMENT OF GASTROINTESTINAL COMPLICATIONS WITH RESION, A MULTIPLE ADSORBENT, IN CHRONIC ALCOHOLIC PATIENTS

MARTIN D. KISSEN, M.D.

H. EDWARD YASKIN, M.D.

and

DAVID R. MORGAN, M.D., Dr. P.H., F.A.C.P.

Philadelphia, Pa.

Chronic alcoholism is a chronic, progressive disease of unknown etiology which represents a major medical and socioeconomic problem. The disease is characterized by an abnormal response to the ingestion of alcohol or alcoholic beverages. The most obvious complications are found in the gastrointestinal system and are the result of irritation produced by the action of alcohol on the gastrointestinal mucosa. The condition progresses to the catarrhal stage involving the whole alimentary tract and producing symptoms of catarrhal gastroenteritis. It must be obvious that this pathologic process leads to suppressed appetite, nausea, vomiting and increased frequency of stools.

There is general agreement that alcohol excites the formation of a gastric juice high in acid and low in pepsin[1]. Observations reported by Beaumont[2] showed that alcohol produces inflammatory changes in the stomach with resultant gastritis. Based on pathologic evidence, Hennings[3] observed that gastritis is frequently associated with alcoholism in man. Dragstedt et al[4] consider the secretagogue effects of alcohol similar to those produced by histamine and suggest that these effects are produced by the liberation of this substance.

An alcoholic patient following an acute exacerbation requires adequate intelligent medical management for his gastrointestinal complications. Various treatments have been suggested and tried; however, none has shown the capacity to control adequately the nausea, vomiting, pains, and diarrhea. The most logical medication for treatment of these symptoms seemed to be a preparation possessing the capacity to modify hyperacidity, to adsorb metabolic and chemical toxins, to relieve pain, and at the same time to allow the assimilation of essential nutrients. A search for a preparation with these attributes led us to Resion,* a polyphasic adsorbent preparation made up of polyamine methylene resin, sodium aluminum silicate, and magnesium aluminum silicate in a palatable vehicle.

From the C. Dudley Saul Clinic for Alcoholism, St. Lukes and Children's Medical Center, Philadelphia, Pa.

*Resion – Supplied by Medical Research Department, National Drug Company, Philadelphia, Pa.

The antacid portion of the formula is represented by the polyamine methylene resin, which was found to possess the additional capacity of adsorbing indole and skatole[5]. Resin was reported to be highly effective in removing bacterial toxins of unknown chemical composition and in removing bacteria themselves[6], in inhibiting lysozyme[7], and in adsorbing tyramine, histamine, putrescine, and cadaverine. A review of the medical literature shows Joslin[8] found Resion very effective in infantile diarrhea; Quintos[9] reported Resion combined with chemotherapy effective in infectious infantile diarrhea; Lichtman[10] successfully treated 200 cases of food poisoning and Fitzpatrick et al[11] found Resion of considerable value in controlling nausea and vomiting associated with pregnancy. The established effectiveness of Resion in the treatment of food poisoning, of nausea and vomiting in pregnancy, and of various types of diarrhea gave us good cause to believe that Resion represented the medication we sought for treatment of the gastrointestinal complications frequently associated with acute alcoholic bouts in chronic alcoholic patients.

We treated 50 consecutive male patients who had gastrointestinal complications following acute alcoholic debauches. Ages ranged from 24 years to 60 years with a large percentage near the mean of 45.

All of the patients on admission had diarrhea manifested by 4 to 10 evacuations daily. About 60 per cent complained of pains, a number referring to them as "belly cramps". Nausea was a complaint in all cases and vomiting in about 50 per cent.

Resion treatment was standardized for all patients in this series. The initial dose was 2 tablespoonfuls followed by a tablespoonful at hourly intervals for 3 doses and then at 3 hour intervals while awake. This treatment was continued in all patients for 3 days, even though improvements were reported earlier.

The gastrointestinal complaints, (pain, nausea, vomiting, and diarrhea) were relieved in 20 patients following the first dose, in 10 patients after the 2nd dose, in 6 patients after the 3rd dose, and in 13 patients within 24 hours. One patient after the first two doses refused further treatment. The appetite was restored in a majority of these patients within 24 hours and in the remaining patients within 36 hours.

Comment

In our small series of 50 cases of gastrointestinal complications resulting from excessive consumption of alcohol, Resion, a multiple adsorbent preparation containing polymine methylene resin, sodium aluminum silicate and magnesium aluminum silicate, proved effective in controlling nausea, vomiting, pain, and in restoring appetite within 36 hours in 49 of these patients.

On the basis of previous experiences with the use of other preparations in the type of case we have discussed, we can state that Resion has given us the best clinical results, and that it merits our recommendations.

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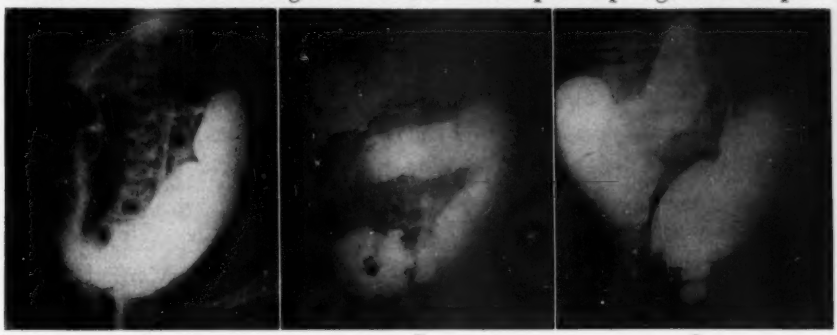
MEGACOLON, VOLVULUS OF SIGMOID, AND HEPATODIAPHRAGMATIC INTERPOSITION OF COLON (CHILAIDITI'S SYMPTOM)

LUDWIG L. FRANK, M.D., F.A.C.P.*

Charleston, W. Va.

Megacolon in infancy and adult life is a rather infrequent but not altogether uncommon disease. Its two relatively most frequent complications besides obstinate constipation, which is the outstanding symptom per se, are volvulus, involving most frequently the sigmoid, and interposition of the transverse colon in the space between the liver and the diaphragm.

It is the intention of this paper to present one case each of congenital megacolon, of volvulus of the sigmoid colon, and of hepatodiaphragmatic interposition. The discussion of these cases will give the opportunity of reviewing the most recent literature pertaining to the subject, and it may stimulate the interest of others in the problems of etiology and therapeutic approach, which both up-to-date are still far from being solved.



A B C

Fig. 1—a) Seventy-two hours after colon-enema, 31 May 1951 (preoperatively). Rectum is empty. Above the sigmoid, the colon becomes abruptly dilated and is filled with barium up to the left flexure. Transverse and right colon are dilated by air. The stomach, greatly dilated by air, is pushed upward into the transverse position.
b) Colon-enema repeated 2 June 1951 (preoperatively), film taken 96 hours after administration of baby enema. In this film the rectum and sigmoid are visualized and the difference in calibre as compared with the descending and transverse colon is less marked, but still present.
c) Colon-enema, 17 November 1951, prone (postoperative).

Case 1:—Congenital Megacolon in a white baby. St. D.P., 3 months old (#48397). Admitted May 30, 1951.

*Gastroenterologist, Staats Hospital, Charleston, West Virginia.

History of constipation since birth. Mother gave daily irrigations to make the bowels move. Gained in weight up to eleven pounds but did not continue to gain during the last weeks. Vomited several times in the last few days.

Pale, dehydrated baby. Abdomen very distended; veins of anterior abdominal wall dilated. Rectum empty. Expelled liquid stool after digital examination of the rectum.

X-ray examination with barium enema: rather markedly dilated colon. The rectum is not as much dilated as the colon proximal to it. Retention of part of the barium enema after 72 hours and 96 hours, respectively, is noted. (Figs. 1a and b).

The child failed to improve and transverse colostomy was done on September 14, 1951. This was followed on October 24, 1951, by the operation for megacolon according to the method advocated by Swenson (Dr. Charles E. Staats). The postoperative course was uneventful and, at the time of writing, the child has greatly improved. This is the more remarkable as re-x-ray on November 17, 1951, 3 weeks after the last operation, still shows marked distention of the entire colon with the most distal segment, which now replaces the resected rectum, markedly narrowed and sharply delineated from the adjacent portion of the generally dilated colon (Fig. 1c).

Comment:—This case fulfills the criteria of true congenital megacolon as postulated by Rosin in 1950:

1. Severe and unremitting constipation since birth,
2. Colonic dilatation,
3. Typical clinical picture.

Rosin, in accordance with others, also asks for hypertrophy of a high degree of the dilated colonic wall. The presence of such a hypertrophy cannot be demonstrated in our case, as the baby so far survived. It is, however, highly probably that it is present.

Roentgenologically the findings were in accordance with the claim of Swenson, 1949, Andressen, 1950, and Bodian, 1951, who see in the demonstration of a narrow bowel distal to the megacolon the decisive point in the roetgenological differentiation between Hirschsprung's disease proper and "idiopathic" megacolon resulting from chronic constipation.

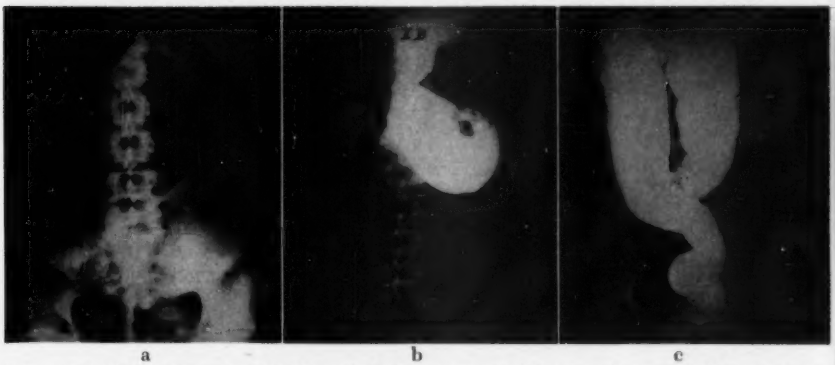
Case 2:—Volvulus of Sigmoid Colon in an adult. Mr. B.N., 50 (#38493). Admitted December 31, 1945.

Constipated as long as he remembers. History of "twisting" of bowels one year before and six months after operation for perforated duodenal ulcer in 1937. Bowels "unlocked" both times under heat applications. Experienced sudden burning pain in abdomen at 5:00 p.m. the day before admission. The pain became

gradually worse and he vomited several times after its onset. Last b.m. at 10:00 a.m. the day before admission.

Acutely and severely ill white male of middle age and in poor state of nutrition. Abdomen greatly distended. Patterning is seen and felt over an oblique area running from the upper left quadrant to the right iliac fossa. Upright film shows huge viscus distended by gas and running balloon-like through the abdomen. Fluid level is seen in left upper quadrant (Fig. 2a).

Immediate operation revealed large roll extending from the left hypochondrium to the right iliac fossa, about 18 inches long, 6 inches wide, firm to pressure, bluish in color, and representing a volvulus which involved the sigmoid. The latter



a b c

Fig. 2—a) Horizontal supine, 31 December 1945. Extreme dilatation of sigmoid. History of previously repaired perforated duodenal ulcer.
b) Prone, 26 February 1946. Preoperative diagnosis: Partial pyloric obstruction following previous operation for perforation.
Emergency operation for volvulus of the sigmoid six months prior to this examination. Large ulcer crater at the middle of the lesser curvature.
c) Barium enema, 22 March 1947, prone. Left colon and sigmoid extremely redundant. Temporary stop in region of upper sigmoid. There is narrowing of diameter in that region. (Picture is reversed).

had sustained a 360 degree rotation about an axis centering on approximately the promontory of the sacrum. The rectosigmoid junction formed the point of obstruction. Release of the obstruction has been obtained by rotating the roll clockwise. The bowel returned to its bed and was anchored to the abdomen wall by a silk suture placed in one of the striae of the sigmoid. Uneventful postoperative course. Two months later, the patient returned complaining again of severe gaseous abdominal distention. X-ray examination on February 26, 1946, revealed 50 per cent retention of the barium meal in the stomach 24 hours p.c., and marked meteorismus of the colon with dilatation of particularly the left colon. The distal portion of the colon was not yet filled with barium at 72 hours p.c. (Fig. 2b).

The stomach complaints persisted and relaparotomy was done in November, 1946, with the preoperative diagnosis of pyloric obstruction. There was definite

evidence of an ulcerating penetrating lesion at the lesser curvature which had caused severe distortion of the entire stomach. Subtotal gastric resection was carried out; the specimen showed a large ulcer crater at the lesser curvature.

Patient continued to suffer from severe acute cramps in lower abdomen. Gastrointestinal tract series studies in March, 1947, revealed extreme meteorismus of the entire colon. Subsequent examination with barium enema showed sigmoid and left colon extremely dilated and redundant. A constant narrowing in the upper sigmoid which to some degree interfered with the free inflow of the contrast was evident. There was accumulation of excessive amounts of gas in the right colon (Fig. 2c).

In April, 1947, sudden, sharp, abdominal pain recurred. On re-admission, a hard, sausage-like roll, ranging through left upper quadrant down to the right iliac fossa was clearly palpable, and there was patterning clearly visible through the distended abdominal wall. Flat plate revealed marked distention of the large bowel and a number of fluid levels. Immediate operation showed the recurring volvulus preoperatively suspected. The redundant colon was picked up, it was only 4 cm. wide at its base. The redundant dilated sigmoidal loop was excised and end-to-end reunion performed.

Postoperative course was uneventful. Patient remained well after the last operation and was symptom-free when last seen in Auust, 1947.

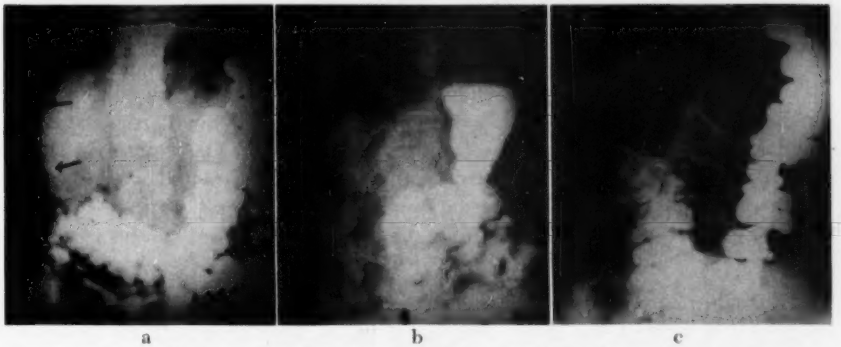
Comment:—This case is remarkable for several reasons. It shows the tendency of the megacolon, and particularly the mega-sigma, to turn around its axis again and again. It furthermore demonstrates the fact that recurrence may occur even after operative disentanglement of the twisted loop and anchoring the sigmoid by a fixating suture. The co-existence of a severe gastric ulcerating lesion is unusual and it is hard to say whether or not this complication or the first laparotomy done on account of perforation of a duodenal ulcer contributed in some way or other to the obstinacy of the volvulus to recur. That it was not the cause of it, is proven by the history of an attack of volvulus one year before the operation for perforation of the duodenal ulcer. Another purely speculative question is why this patient acquired a penetrating ulcer at the lesser curvature of the stomach subsequent to the formerly repaired duodenal ulcer. And finally it should be noted that the patient not only survived but was cured in spite of the multiple operations which were necessary for the ulcers of the duodenum and the stomach on the one hand, and for the recurring volvulus of the sigmoid on the other. This is the more gratifying when we read in the literature that in volvulus alone the operative mortality reported by clinics employing operative treatment of volvulus as the treatment of choice is as high as 40 to 60 per cent (Brunsgaard, 1947).

Case 3:—Hepatodiaphragmatic Interposition of the Colon. Mr. H. A. D., 48 (#37240). The patient was first seen in July, 1942.

Severe constipation experienced for many years, gradually getting worse. Tremor of right hand and arm, numbness in right leg, no disturbance of reflexes. Gastrointestinal tract series (July, 1942): Subdiaphragmatic interposition of colon, redundancy of sigmoid, marked colostasis (Fig. 3a). Barium enema (July, 1942): Balloon-like meteorismus of both flexures, pronounced redundancy of sigmoid. Interposition not seen with the barium enema.

Gastrointestinal tract series repeated February, 1943: Interposition by what seemed to be the unusually long redundant sigmoid seen again (Fig. 3b).

Mental condition deteriorated in the subsequent years and the constipation persisted. Symptoms of Parkinsonism steadily progressing in severity. Barium enema July, 1946, showed the extreme redundancy of the left colon, already found on previous examination. However, the interposition between liver and diaphragm



a b c

Fig. 3—a) Prone 7¾ hours after barium meal (19 July 1942). The interposition of a long segment of air-filled colon between liver and right diaphragm is seen.
b) Prone after barium meal (1 February 1943). The interposition of an air-filled segment of colon between liver and right diaphragm is even more conspicuous than in a. The haustration of the interposed colon is well seen in the original film.
c) The proximal colon is greatly dilated by air. The interposition between the liver and diaphragm is not seen any more.

was not seen on this occasion. Abdomen always distended. No b.m. at times for a whole week despite laxatives and enemas.

Gastrointestinal tract series repeated August 20, 1947, did not show the interposition, but the barium was retained for 9 days. The lower descending and the sigmoid colon showed extreme redundancy and marked dilatation. These colon segments at this time were seen to be displaced into the upper abdomen, running along the edge of the liver to the right abdomen and then descending to the rectum (Fig. 3c).

Together with the severe and persistent constipation the Parkinson symptoms went from bad to worse, the entire personality deteriorated, and complete physical and mental helplessness set in.

He was taken home and died shortly afterward.

Comment:—The opportunity of observing a case of interposition of the colon between the liver and the diaphragm is rarely given, and the literature dealing with this condition is accordingly sparse (Bockus, 1944).

In the discussion of its etiology three groups of factors are to be taken into consideration:

1. Hepatic
2. Diaphragmatic
3. Intestinal

(Choussat Chaussee, 1937, quoted by Bockus). Of these groups, the most important one probably is the third, which accuses in the first place the abnormality known as megacolon for the incidence of the interposition.

Hurst saw 5 such cases in the material of megacolon studied by him (Hurst, 1944).

The syndrome under discussion has been described first by Chilaiditi in 1910 (quoted by Rubacher, 1946). Chilaiditi, for whom the condition carries its name (Chilaiditi Symptom), blamed the liver, its faulty position (hepatoptosis) or its defective suspension, for its occurrence. Rubacher (l.c.) quoted a number of authors, who in the years following the publication of Chilaiditi's published opinions about the etiology which were quite different from that in Chilaiditi's original paper.

Rubacher himself presented as his conviction the thesis that the symptom is congenital in most of the cases. This would be in accordance with the observations of Hurst (l.c.), who in his publications connected the syndrome of Chilaiditi with the syndrome of congenital megacolon, known in literature as Hirschsprung's disease (1888).

Discussion

The interest in megacolon and its allied conditions has been reawakened in recent years. It is now recognized that its incidence is not restricted to infancy, but may be encountered even more often in adult life (Hurst, 1944, l.c.).

The differentiation between the congenital and acquired megacolon is still somewhat hazy if not impossible in part of the cases (Swartz, 1947). It is definitely conflicting, in so far as some writers claim that in congenital megacolon part or all of the colon, and the rectum as well, are dilated and hypertrophied (Hurst, 1944, l.c.; Rosin, 1950), whereas others, mostly from roentgenological evidence, stress the point that in congenital megacolon the rectum does not participate in the dilatation of the colon proper (Swenson, 1949; Andressen, 1950).

The uncertainty which exists in the possibility of strict differentiation between the true congenital megacolon and "idiopathic" or acquired megacolon has

led some writers to the compromise of abandoning this differentiation altogether, and to summarize the cases of megacolon in three topical groups irrespective of the question as to whether or not the rectum is involved (Grimson, 1944).

The difficulty and sometimes impossibility of clean separation of the congenital from the acquired form of megacolon is closely interrelated with the not yet conclusively settled problem of the pathogenesis of either. The theory of Hurst advocates the failure of the rectal sphincter to relax (achalasia), and substantiates this theory by anatomically demonstrated destruction of the nerve elements of the Auerbach plexus in the region of the rectal sphincters (Hurst, 1944, l.c.).

On the basis of his theory he attempts to draw a clear line between congenital and acquired megacolon, ascribing the first type to paralysis of the parasympathetic nerve elements in the rectal wall due to inflammation, atrophy, or fibrosis, and the second to dyschezia which is believed to be due to "disease" of Auerbach's plexus with permanent increased tone of the lower rectal sphincters. Bodian, 1950, and Hiatt, 1951, accept this theory and believe that there is a "lack of propulsive capacity of the rectum, or rectum and sigmoid, apparently owing to insufficient number of parasympathetic ganglion cells in the wall of the distal rectosigmoidal segment". Best and Taylor, 1950, however, do not agree with this hypothesis and believe, on the contrary, that "the motor-mechanism is intact but under the influence of an inordinate inhibitory action exerted through the sympathetic."

The far-reaching consequences of such a divergence of opinions can only be mentioned here. These consequences immediately become apparent when the most modern trends of treating the condition by surgical means are contrasted. The surgical procedures now recommended for curing severe cases of true megacolon, or its most important complications in infants and in adults as well, are quite different ones, and their rationale is determined by the respective ideas of the authors in regard to the basic etiology of the condition. They are in short abstract as follows:

One stage resection of the megacolon and ileosigmoidotomy in three cases of acute emergency, two of them with volvulus of the sigmoid (Grimson, 1947).

Sympathectomy supplementary to medical treatment, with rather discouraging results (Grimson, 1944).

Excessive sympathectomy alone in two adolescents with Hirschsprung's disease, with good results in both cases (Gelderen, 1946).

Exteriorization and second stage resection in volvulus of the sigmoid colon (Griffin et al, 1945).

Wangensteen method of primary resection and anastomosis, or a four-stage method (cecostomy, Mikulicz exteriorization resection, closure of the sigmoidostomy, closure of the cecostomy) as interval operation in carefully selected cases

of volvulus of the sigmoid which have undergone repeated torsion (Brunsgaard, 1947).

Colectomy in operative cases of primary megacolon as procedure of choice; experiences with sympathectomy alone discouraging (Swartz, 1947).

Partial bilateral sphincterectomy in congenital megacolon in children, eventually in combination with other surgical procedures (J. A. Jenkins, 1948).

Removal of the distal narrowed malfunctioning segment in congenital megacolon (Swenson, O. et al, 1949; M. Andressen and B. Kromann, 1950; M. Bodian et al, 1950; 1951; Hiatt, 1951).

It becomes evident from this compilation that the surgical approach in Hirschsprung's disease has shifted more and more to attacking the rectosigmoidal segment, somewhat along the lines of Hurst's conception, but leaving in suspense whether the vagal or the sympathetic part of the innervation of the rectosigmoidal segment is at fault.

It is the concern of the surgeon to pick up the appropriate operative method in those cases of Hirschsprung's disease which really are in need of surgical approach. The majority of cases don't, and proper medical management in these non-operative cases has excellent results (Swartz, 1947). Its essential principles are low residue diet, regular enemas, no laxatives; additional use of antispasmodics and sedatives if indicated.

If volvulus occurs in a case of megacolon, the case under treatment has to be treated individually. Procedures like administration of a long rectal tube through the proctoscope into the strictured area (Brunsgaard, 1947) may be tried, but no time should be unduly lost before operation is decided upon. Our own experience in the case reported and the experience of others (Griffin, 1945) point strictly to the opinion that in cases not complicated by gangrene, simple detorsion is not satisfactory and primary resection with the one or the two stage method is the operation of choice.

Interposition of the colon between liver and diaphragm as a sequela of megacolon is of casuistic-roentgenological rather than surgical interest. Medical management usually will suffice in such cases, particularly if they are, as in the case presented, of the sliding type (Bockus, 1944), and provided that no other complications like volvulus have arisen. Mention should be made in this connection of the fact that the differentiation between congenital and acquired megacolon is not of purely academic value. A very recent report stresses the high incidence of acquired megacolon in neurological disorders, especially Parkinsonism (Lewitan et al, 1951). Our own case is an example of that. Lewitan and his co-workers are definitely right in warning in such cases against the long-standing use of parasympathetic paralytic drugs such as hyoscine, atropine, and other belladonna derivatives, and against unnecessary operations as well.

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EDITORIAL

The Bile in Pregnancy

The composition of bile undergoes a change during pregnancy and the cholesterol content increases, however, toward the end of gestation the bile contains less cholesterol. It has been found that the cholesterol rises after the third or fourth day pospartum. Significantly, the cholesterol level is higher in nonlactating women. Whether or not this change in the composition of the bile would tend toward formation of gallstones is still a moot question.

Cholecystography after egg-yolk mixture showed that there is marked delay in gallbladder evacuation during pregnancy. At times emptying occurs only after six hours. Following atropine injection the gallbladder may empty in two hours. Shafer assumes that a modification of the vegetative nervous system during gestation, brings about a decrease in gallbladder function; stasis results and this has been termed a hypertonic gallbladder. Potter reported that the gallbladder was abnormally enlarged in 75 per cent of 390 women delivered by cesarian section, and in two per cent of these patients culture of the bile showed bacteria. S. Weiss states that pregnancy is a strong predisposing factor. It may be that the enlarged uterus crowds the viscera and exerts pressure on the common duct, thus obstructing the flow of bile.

Abnormal secretion of the endocrine glands during gestation may bring about an increased cholesterol content in blood. Another consideration is the fact that in pregnancy there is a functional disturbance in liver activity resulting in an underproduction of bile salts which directly influence cholesterol metabolism. A reduced amount of bile salts will precipitate cholesterol crystals in the gallbladder.

Kiegel and his associates made studies of gallbladder function in pregnancy. Thirty-four specimens of bile removed at term during the course of cesarian section showed an increase in cholesterol concentration, while bile salt concentration remained unchanged, or slightly decreased.

It is axiomatic that the highest incidence of gallbladder disease and especially cholelithiasis occurs in multipara. The beginning of biliary stasis has frequently been demonstrated radiologically during the child-bearing period, by delayed evacuation of the gallbladder after fatty meals. Moreover the cholesterol and bilirubin content of the blood of the expectant mother is usually high. Hepatic function tests occasionally show liver insufficiency.

These facts should direct the attention of the obstetrician and prenatal clinic staff to the need of providing prophylactic measures against later gallbladder disease. If a liberal fruit and vegetable diet together with a daily dose of mineral

oil, do not correct constipation, then a mild saline cathartic should be given at breakfast time. This will serve the purpose of stimulating gallbladder activity.

Abnormally high blood cholesterol values call for dietary and medical therapy. If there is evidence of biliary stasis, duodenal bile drainage is indicated. For some time postpartum the biliary tract should be carefully watched for signs of impending disturbances. It is much simpler to correct the condition in its incipient stage, than later when organic changes in the gallbladder have developed.

BERNARD WEISS

ABSTRACTS

PATHOLOGY AND LABORATORY RESEARCH

A NEW DYE (BROMPHENOLBLUE) FOR LIVER FUNCTION TEST: Isamu Kaito, Sigemiti Tuda, Tamotu Numasawa and Tatuo Nakazima. Tohoku J. Exper. Med. 54: No. 4 (Oct. 25), 1951.

Dyes for liver function tests have been investigated by many authors and the excretion of the dye into the feces, bile, urine and retention in the blood steam have been tested clinicaly. Nakagawa reported that a dye which is eliminated only in the bile and not in the urine is most advisable for testing its retention in the blood stream, while for testing the urine, it is preferable that a greater part of the dye appears in the bile and only a small proportion in the urine in normal conditions, and that an increased output and prolonged duration of the excretion in the urine are noticed when a disturbance of elimination occurs. In this paper the authors report bromphenolblue with a pH of 4.6 which can be applied to testing the urine in a similar way to azorubin S. Its solution is obtained by adding 1.5 c.c. of 0.1 N NaOH per 100 mg.

The elimination of the dye in the urine and bile was investigated in the dog. The urine is catheterized by the method introduced by McMaster and Elman. Three c.c. of 0.5 or 1.0 per cent solution is injected into the large saphenous vein of a dog and collected every 30 minutes until the dye disappears. The percentage readings of excretion for injected amount are estimated. The total excretion in the urine was 7.4 and 5.1 per cent with 0.5 and 1.0 per cent solutions respectively. The time required for first appearance in the urine was 5 minutes and the total excretion showed 12.4 per cent. We found more of an increase in output of bromphenolblue in the bile than with azorubin S and the contrary result in the urine, showing that the total output of bromphenolblue in bile and urine is more than that of azorubin S.

On the other hand it is confirmed that bromphenolblue never appears in the gastrointestinal juice. Even a trace of the color could not be found in the gastric and intestinal contents by postmortem examination one hour after injection into the blood stream of a dog whose common duct was ligated. After intravenous injections fractional collections of gastric juice in man for 2 hours by means of the Rehfuss tube revealed no dye.

The authors also observed the elimination in urine before and after experimental disturbance of the liver in a dog. The total excretion after giving 3.0 c.c. of 0.5 per cent solution intravenously showed 8.8 and 8.5 per cent before damage. While it increased remarkably showing 35.4 per cent one day, 54.1 per cent three days after subcutaneous injection of 1.0 c.c. of chloroform per Kg. Twelve days after injections it decreased to 8.1 per cent.

Four c.c. of 0.5 per cent solution is administered intravenously and the urine is collected hourly for the first 5 hours, and later every 2 hours until the disappearance of the dye from the urine. The percentage of excretion for the injected amount is obtained by measuring each specimen and estimating the amount of dye excreted calorimetrically. In normal persons the excretion was all below 12.0 per cent and the time required for disappearance was less than 5 hours. Ten out of 12 cases of liver and gallbladder diseases indicated excretions over the normal reading; 3 cases of hepatitis and one case of gall stones showed a marked increased excretion of over 50 per cent.

Conclusions: 1) Bromphenol blue appears in the bile in a very high concentration and in small proportions in the urine. Its elimination in the urine increases in liver disturbances.

2) This dye is one of the most suitable for the liver function test estimated by means of the excretion in the urine.

SHIGETADA KODAMA

PROTEOLYTIC ACTIVITY OF HUMAN GASTRIC MUCOSA: Mitsuhisa Hayakawa. Tohoku J. Exper. Med 53:251 (March), 1951.

In human gastric mucosa tryptase activity is surely present, but against it serum albumin and egg albumin are usually resistant. Tryptase of human gastric mucosa may partially find itself in desmo-form and its activity remains unaffected also in the presence of nondialyzed serum albumin or egg albumin. The hydrolysis of gelatin seems to

undergo some retardation in the presence of human serum but not that of casein. The tryptase activity is rather inhibited by cystein, but no effect of kinase activiation is found. Tryptase activity of human gastric mucosa undergoes activation in the presence of dialyzed albumin, with which such a protein as egg albumin comes to be hydrolyzed, though it is usually resistant to tryptase activity.

FRANZ J. LUST

LIVER AND BILARY TRACT

VALUE OF PREOPERATIVE MANOMETRIC AND ROENTGENOGRAPHIC EXAMINATION IN THE DIAGNOSIS OF PATHOLOGIC CHANGES AND FUNCTIONAL DISTURBANCES OF THE BILIARY TRACT: P. Mallet-Guy. Surg. Gynec. & Obst. 94:385-393 (Apr.), 1952.

A technic of preoperative manometric and roentgenographic examination of the biliary tract is presented. Between 1942 and September 26, 1951, fifteen hundred operations on patients have been performed, as well as extensive experiments on dogs.

The technic consists of two successive and independent steps: A manometric recording and a cholangiographic examination. The examination should be as simple, short, and standardized as possible.

Performed during a cholecystectomy, it demonstrates diverse pathological conditions which are factors in postoperative recurrences. After removal of stones in the common duct, it is possible to show that no other obstruction remains and to decide whether external drainage, primary suture, or a choledochoduodenostomy is required. Manometric and roentgenographic examination for malignant obstruction of the common duct and for biliary functional disturbances by the method of puncture of the gallbladder are described.

J. R. VAN DYNE

CHOLEDOCHOSTOMY WITH CHOLANGOGRAPHY: M. Corff, S. Berger, and J. Gershon-Cohen. Surg. Gynec. & Obst. 94:394-400 (Apr.), 1952.

The cases of fifty patients who had choledochostomies and one or more cholangiograms done are reviewed.

It was found: 1. That there was no mortality in this group. 2. Types of operation in addition to the choledochostomy were given and that 80 per cent of these were cholecystectomy. 3. The indications for the common duct exploration were noted, and the most frequent one found to be jaundice or a history of jaundice. 4. There was a breakdown given of the causes of the jaundice, the greatest number (61 per cent) of which were due to stones in the common duct. 5. The types and numbers of the cholangiograms were discussed and the most logical one (through the cystic duct stump) was commented on in great detail. 6. Retained stone in the common duct (18 per cent) was discussed. 7. Suggestions for the prevention of retained stones were made.

J. R. VAN DYNE

ACUTE GASEOUS CHOLECYSTITIS: B. R. Mooney and K. E. Matzinger. Canad. M. A. J. 66:66 (Jan.), 1952.

Acute cholecystitis with gas present in the lumen of the gallbladder is due to infection with gas forming microorganisms. The condition is rare. There are 23 cases reported in the literature. The majority occur in the sixth and seventh decades. Of the 23 cases 8 were diabetics, suggesting the possibility that arteriosclerosis and the tendency to thrombosis found in diabetes might play a role in the etiology.

The first case diagnosed from an x-ray plate was reported in 1931 by Hegner.

The etiological agents were Clostridium Welchii, Escherichia coli and anerobic streptococci and staphylococci.

The gallbladder was distended with foul smelling exudate mixed with gas bubbles. Gas was found in the submucosa causing a complete separation of the mucosa from the muscularis. Gallstones are often present. Per-

icholecystitis with massive adhesions was usual.

The clinical course is not unlike that of acute cholecystitis.

X-ray signs are pathognomonic. In early stage, gas is seen in lumen of gallbladder and a fluid level can be seen in films taken in the upright. The gallbladder is enlarged. Later gas appears in the submucosa along the entire wall of the organ. Still later gas disappears from the lumen of the gallbladder and the layer of gas in this sub-mucosa is slowly absorbed. The gallbladder decreases in size after several weeks.

Surgery in acute cases is contraindicated unless there is perforation.

Conservation theory is the treatment of choice.

JOSEPH SCHWAB

PORTACAVAL SHUNTING FOR PORTAL HYPERTENSION: A. H. Blakemore. Surg. Gynec. & Obst. 94:443-454 (Apr.), 1952.

In reporting on 160 cases of portal hypertension in whom portacaval shunts were established, evidence was presented correlating the reduction in portal pressure with the reduction in size of esophageal varices and the prevention of hemorrhage. Evidence has accumulated from analysis of these patients with portal hypertension in whom shunts were established, that the primary role of portacaval shunting is:

1. To protect patients from untimely death due to hemorrhage; 2. To protect livers in cirrhosis cases from the deleterious effects of recurring hemorrhage and wasting ascites thus affording an opportunity for healing and regeneration of liver parenchyma under the favorable influence of modern medical management.

J. R. VAN DYNE

GALLSTONE ILEUS: P. Nemir. Surg. Gynec. & Obst. 94:469-475 (Apr.), 1952.

Eight cases of gallstones ileus occurring between 1939 and 1951 in the hospital of the University of Pennsylvania are presented. One of these patients died. Gallstone ileus should be strongly suspected in an elderly obese person presenting the signs and symptoms of intermittent recurrent obstruction progressing to complete obstruction and in which a history of gallbladder disease is obtained. The high mortality which occurred in this group of patients was the result of age, obesity, associated complicating conditions, too extensive surgery, and, most important, delay in diagnosis. If the physician is kept constantly aware of the occurrence of gallstone ileus with a result in decrease in the time lapse between onset of symptoms and institution of active treatment, then it is believed, the mortality will be appreciably decreased.

J. R. VAN DYNE

BOOK REVIEWS

ROENTGEN EXAMINATION IN ACUTE ABDOMINAL DISEASES: J. Frimann-Dahl, M.D., Ph.D., Chief of Roentgen Department, Ulleval Hospital, Oslo, Norway. 323 pages. 357 illustrations. Charles C. Thomas, Publishers, Springfield, Ill., 1951. Price $10.50.

Here is a book that should be on the desk of every physician. It is written by one of the foremost roentgenologists. Owen H. Wangensteen wrote the foreword and he states: "The patient's life may depend on the course of action pointed up by a judicious assessment of clinical and x-ray findings."

Jack Friedman includes a chapter on the use of the Miller-Abbott tube.

The author stresses that all pathological cases found in this book have been verified either at operation, necroscopy or by the clinical course.

The illustrations, text, references and cross-index are concise and comprehensive. It is recommended as a must.

ANTIBIOTIC THERAPY: Henry Welch, PhD. and Charles N. Lewis, M.D. with a foreword by Chester S. Keefer, M.D. 562 pages with extensive references and cross-index. The Arundel Press, Inc., Washington, D.C., 1951. Price $10.00.

Antibiotic therapy has been used by a great many physicians without full knowledge of its power. Many have prescribed it in a hit or miss manner and at times with dire results. This well printed and informative volume should be a guide to the doctor who uses antibiotic preparations in private and/or hospital practice.

In reading this interesting and informative volume, the reviewer has now a clearer concept of when, how and what preparation will be useful in a given case.

Every physician is urged to get "Antibiotic Therapy" and keep it handy as a reference.

PRINCIPLES OF INTERNAL MEDICINE: Edited by T. R. Harrison, M.D., M. M. Wintrobe, M.D., Paul B. Beeson, M.D., William H. Resnick, M.D. and George W. Thorn, M.D. 1590 pages with numerous tables, diagrams and illustrations. The Blakiston Co., Philadelphia, Pa., 1950. Price $12.00.

Books on medicine have been written by many clinicians, but Harris' book outshines them all. This large volume is divided into seven parts and each part is divided into numerous sections. For instance, Part I deals with Cardinal Manifestations of Disease and this is divided into eight sections. Each section written by a master clinician and followed by selected references.

Students and general practitioners will find a wealth of information between the covers of this tremendous volume. Physicians in the several specialties will also do well to peruse these pages and undoubtedly will enhance their knowledge.

Physicians preparing to take examinations given by the specialty boards and senior medical students taking their final examination or the boards for licensing, should unhesitatingly devote time studying the new material found in this text.

CLINICAL LABORATORY METHODS: W. E. Bray, B.A., M.D., Professor of Clinical Pathology, University of Virginia, Director of Clinical Laboratories, University of Virginia Hospital. Fourth Edition. 119 text illustrations and 18 color plates. 614 pages. The C. V. Mosby Co., St. Louis, Mo., 1951. Price $7.25.

This fourth edition of a very useful bookpathologists. Informative new illustrations has been brought up-to-date by the additionand charts have been added and some secof procedures recently described by clinicaltions have been rewritten and enlarged.

This book is an excellent "companion-addition" to the desk library of physicians generally and for clinicians and laboratory workers and should be very helpful to internes, residents and candidates preparing for specialty board examinations. It may be added on the shelf, placed alongside of other books of a similar nature put out by the same publisher.

METABOLIC METHODS — CLINICAL PROCEDURES IN THE STUDY OF METABOLIC FUNCTIONS: C. Frank Consolazio, Chief of Biochemistry, U. S. Army, Medical Nutrition Laboratory, Chicago, Ill., Robert E. Johnson, M.D., Ph.D. (Oxford), Professor and Head of the Department of Physiology, University of Illinois, Urbana, Ill. and Evelyn Marek, M.A., Biochemist, U. S. Army, Medical Nutrition Laboratory, Chicago, Ill. Illustrated. 471 pages. The C. V. Mosby Co., St. Louis, Mo., 1951. Price $6.75.

This work by experts in physiology, biochemistry and nutrition presents in detail proved methods found useful by the experienced authors during 15 years of research in the field of mammalian metabolism, particularly that of human beings in health and in disease.

This book is enthusiastically recommended to students, undergraduate and postgraduate, internes, residents, general practitioners and clinicians interested in metabolic methods of study.

PHYSICAL DIAGNOSIS: Raymond W. Brust, A.B., M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania Medical School. Introduction by Truman G. Schnabel, A.B., M.D., F.A.C.P. 294 pages. Appleton-Century-Crofts, Inc., New York, N. Y., 1951. Price $5.00.

The author of this very practical little book on *physicial diagnosis* will be of great help to all medical students and the book will be a "refresher" work for internes, residents and general practitioners.

The author received his training and encouragement from the late Dr. David Riesman and from Drs. Truman G. Schnabel and David N. Kremer of the Philadelphia General Hospital.

The methods of examination are those taught to medical students at the University of Pennsylvania.

The author has been engaged in preceptorial guidance to these students for 20 years and this little book contains informatively and instructively arranged material, that is most useful to all medical students.

SURGICAL CARE — A PRACTICAL PHYSIOLOGIC GUIDE: Robert Elman, M.D., F.A.C.S., Professor of Clinical Surgery, Washington University, School of Medicine, Assistant Surgeon, Barnes Hospital, Associate Surgeon, St. Louis Children's Hospital, Director of Surgical Service, H. G. Phillips Hospital, St. Louis, Mo. 586 pages. Appleton-Century-Crofts, Inc., New York, N. Y., 1951. Price $10.00.

This book on "Surgical Care" by an experienced surgeon is truly a practical "Physiologic Guide".

The author gives brief, interesting "historical surveys", discusses very briefly the adrenocortical hormones, adrenocortical response and the use of adrenocortical extracts, changes in protein, carbohydrate and salt metabolism, the surgeon and the internist and the psychiatrist, nutrition in surgery, water and salt depletion, protein deficiencies, vitamin deficiencies, subcutaneous infection, intravenous infusion, preoperative care and laboratory examinations, correction of deficits, cardiovascular disease, hepatic disease and diabetes, anesthesia, cardiac resuscitation, postoperative care, chemotherapy in surgery, pulmonary edema, atelectasis and abscess, thromboembolic disease, pulmonary embolism, anticoagulant therapy, renal impairment, surgical shock, hemorrhage, gastric atony, abdominal distention and acute gastric dilatation paralytic ileus, peritonitis and thermal burns.

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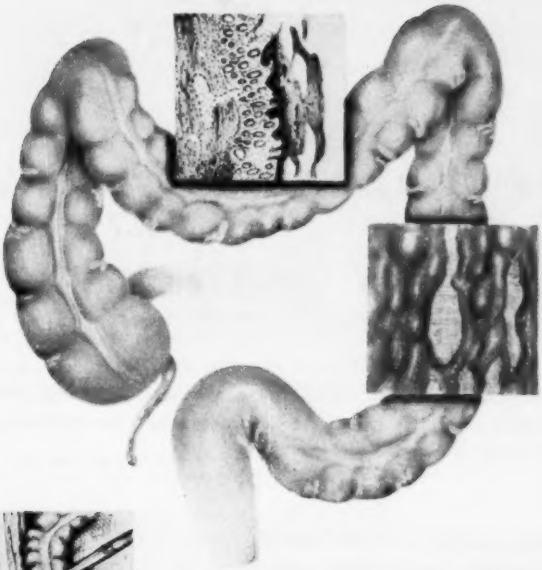

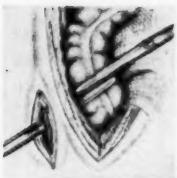

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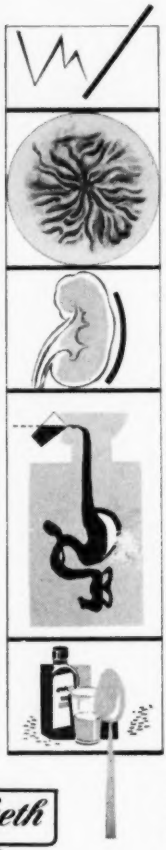

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